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Abstract

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Instability in an SIR-model with age-dependent susceptibility

This text consists of two papers on age-structured epidemic models of the SIR-type. Both papers utilize the observation that for most diseases the infectious period is much shorter than the average life span of the hosts.

In the first paper, *The effect of age-dependent host mortality on the dynamics of and endemic disease,* I show that when disease transmission is independent of age, the endemic equilibrium is always stable. I discuss in some detail how the dominant eigenvalues depend on the distribution of host life lengths.

The second paper, *Instability in an SIR-model with age-dependent susceptibility,* deals with the situation where disease transmission depends on age. I give conditions for the stability of the endemic equilibrium and show how the endemic equilibrium may loose its stability through a Hopf-type bifurcation.

The effect of age-dependent host mortality on the dynamics of and endemic disease is in press in Math. Biosci.

Instability in an SIR-model with age-dependent susceptibility is submitted to Proc. 3rd Int. Conf. Math. Popul. Dynamics, Pau, June 1992.

The effect of age-dependent host mortality on the dynamics of an endemic disease

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Abstract: Using asymptotic expansions in the ratio between the duration of infection and host life length, I analyze equilibrium conditions for an SIR-type epidemic model with age-dependent mortality and age-independent disease transmission. Disease incidence at equilibrium depends on the distribution of life lengths. Incidence is maximal if host life span is fixed and — for vanishing higher moments — it decreases with increasing variance of the distribution. The spectrum of the linearization about the endemic equilibrium has two dominant components, one near 0 and one with large imaginary part. All roots have negative real part so the model is always stable. The roots with large imaginary part dominate in most cases, indicating that the approach to equilibrium will be through slowly damped oscillations.

The purpose of this paper is to study the effect of host demographic structure on the dynamics of an endemic disease under the simplest possible assumptions about disease course and transmission. Classical models of epidemiology assume a constant mortality and hence an exponentially distributed duration of life. In contrast I here allow host life-lengths to follow a general distribution with some restrictions in variation and range leading to a more realistic demographic structure. The vital dynamics of the host population affect disease transmission. If exposed individuals acquire permanent immunity, older individuals are more likely to be immune due to previous exposure [20]. Therefore, in a population where mortality increases with age, more deaths will occur among immune individuals than in a population with constant mortality. Our aim is to understand the effect of the mortality structure on the dynamics near the endemic state.

Table 1. Duration of infectiousness D and reproduction ratio R_0 for some viral diseases. The reproduction ratio varies with population density and social conditions. The ratio ϵ of D to average host life span A is computed for $A = 71.6$ years. Data for influenza from [40] and [9]; all other data from [1].

	$D = \nu^{-1}$ in days	$\epsilon = D/A$	R_0
Measles	6–7	$2.3–2.7 \times 10^{-4}$	5–16
Chicken pox	10–11	$3.9–4.2 \times 10^{-4}$	7–10
Rubella	11–12	$4.2–4.6 \times 10^{-4}$	6–7
Influenza	2–3	$0.8–1.2 \times 10^{-4}$	2–5

Recently, age-structured models of epidemics have been studied intensively for both biological and mathematical reasons. Biologically the host age-structure is important for several aspects of infectious disease epidemiology: including age-dependent disease transmission [2, 4, 37]; age-dependent severity of infection [3, 21, 31]; and age-stratification of empirical data [8, 20]. In this study we isolate the effects of age-dependent host mortality, thus providing a baseline to which one can compare the effects of additional age-dependent factors.

Parallel to the biological interest, age-structured models have received much attention in the mathematical literature starting with the work of Hoppensteadt [28] and Dietz [20]. By now it is well known that age-dependent SIS- and SIR-models are well posed for reasonable choices of age-dependent parameters. In addition, Dietz and Schenzle [22] identified a threshold quantity, the reproduction ratio, that determines the existence of an endemic equilibrium and the local stability of the disease-free equilibrium [12, 16, 24, 25]. Busenberg et al. [12] show that in an SIS model the endemic equilibrium is always stable when it exists, while Thieme [41] recently found conditions for the destabilization of the endemic equilibrium in an SIR-model. The results are formulated also in a modern functional analytical frame allowing for some global results and a more general formulation of the threshold and stability conditions [14, 19, 29].

Clearly the general question of the local stability of the endemic equilibrium is hard since the characteristic equation is transcendental and quite complicated. Our focus will be on deriving asymptotic expressions for the spectrum of the linearization around the endemic equilibrium. To simplify the computations, I focus on realistic parameter values. Table 1 shows that for many viral diseases, the system involves processes at two different time scales, namely the host death and renewal processes and the duration of infection. The ratio between the two time scales can be 3–4 orders of magnitude thus allowing for simplification by the use of asymptotic expansions [6].

Inaba [29] and Thieme [41] report that the endemic equilibrium is stable at low disease levels for a model with age-dependent infectivity and mortality. Their proof consists in studying the limit of the spectrum as the force of infection goes to zero. From biological reasoning, I will argue that one should rather fix the force of infection and study the behavior as ε the ratio of the two time scales goes to zero. My analysis of will show that in this limit the dominant part of the spectrum is determined by a stability equation containing a singularity that leads to two types of roots. One group of roots lies near the origin while a pair of roots have imaginary part on the order of $\varepsilon^{-1/2}$ and real part of order unity. Both types of roots remain in the negative half plane, showing that the endemic equilibrium is always stable. The roots with large imaginary part are associated with the slowly damped oscillations characteristic of epidemic models. However it is not clear that these roots always dominate.

The presence of multiple time scales has been recognized in connection with numerical difficulties [16], and Anderson and May [3, 5, 33] use first order expansions to give approximate expressions for the age-dependent disease incidence and a discretization in time to asses the period of the oscillations in models similar to the one we study here. Numerical studies show that the endemic equilibrium is stable with slowly damped oscillations, e. g. [30] and I here give an explanation for these observations.

On the short time scale several processes may not be well approximated by simple rates as is used in SIR-models. Time of recovery is not exponentially distributed e.g. [9] and Tuljapurkar and John [43] find by incorporating the daily pattern of human contacts that the force of infection may be non-linear especially in sparsely populated areas. Since our concern is with the long time

scale, we neglect these details; in particular we will use standard estimates of the transmission potential R_0 to access the magnitude of the force of infection.

Since mean life length is a key aspect, the age-dependent mortality is not a convenient way to describe the host demographic structure. In stead I use the distribution of host life-lengths and its mean value. Thieme [41] transforms in a similar manner using the distribution of the age of infectives and obtains quite similar expressions for the stability equation, but his stability equation is not amendable for the limit procedure we wish to apply. In my formulation the model becomes similar to distributed delay models and the stability analysis will utilize methods from that field [10, 17, 32].

In the first part of the paper, I state the age-structured version of the SIR-model with age-dependent mortality and demonstrate by rescaling how ε, the ratio between the two time scales, enter. In order to determine the stability properties of the model, we will need second order approximations of the Lotka characteristic equation. As a first step, I analyze the endemic equilibrium and obtain an expansion in ε of the force of infection at equilibrium. The machinery is now set up for a description the dominant part of the spectrum of the linearization. Finally I study in more detail gamma distributed life-lengths and show that in this cases the roots with large imaginary part dominate.

1 Multiple time scales

Our starting point is the well known age-dependent SIR model of the transmission dynamics of an immunizing contagious disease in a host population where we take into account the host's vital dynamics. For a detailed derivation see [28]. With respect to the disease, the host population is divided into 3 classes, susceptibles S, infectious I, and recovered and immune R. Each of the classes is further divided according to host age a so that $S(a,t), I(a,t)$, and $R(a,t)$ denote the age distribution of susceptibles, infectious, and recovered respectively. Hence $\int_a^b S(\alpha,t)\,d\alpha$ gives the number of susceptibles between age a and age b at time t, etc. For simplicity we will assume that the rate of recovery ν is a constant independent of age and time since infection and that disease transmission is independent of age, so that the force of infection Λ is proportional to the number of infectious. Some of our results will hold for

age-dependent infectivity and we will return to this question in the discussion. With these assumptions we have

$$\begin{aligned}
\frac{\partial S}{\partial a}+\frac{\partial S}{\partial t} &= -\Lambda S - m(a)S \\
\frac{\partial I}{\partial a}+\frac{\partial I}{\partial t} &= \Lambda S - \nu I - m(a)I \\
\frac{\partial R}{\partial a}+\frac{\partial R}{\partial t} &= \nu I - m(a)R \qquad (1) \\
\Lambda(t) &= \beta \int_0^\infty I\, d\alpha \\
S(0,t) &= \varrho \qquad I(0,t) = R(0,t) = 0.
\end{aligned}$$

New individuals are born susceptible, and the birth rate ϱ is constant, insuring that the total population size is fixed:

$$\int_0^\infty (S+I+R)\, d\alpha = \varrho \int_0^\infty e^{-\int_0^a m(\alpha)\, d\alpha}\, da = N.$$

The equation for R is thus redundant. Since we are concerned with the limit behavior, we omit explicit reference to initial conditions. As is common when modeling the spread of a directly transmitted disease, the incidence rate βSI is assumed to be proportional to as well the number of infectious as to the number of susceptible [1]. For large populations it is biologically more reasonable to assume that the incidence rate is proportional to the fraction of individuals who are infectious [39], but since we are concerned solely with models of fixed population size N this will lead to the same basic model.

To simplify the analysis we observe that model (1) encompasses two different time scales, since the host renewal process is associated with the mean host life-span A while the recovery process is associated with the duration of the disease $D = \nu^{-1}$.

The effect of the two time scales becomes clear after rescaling time t and age a in units of host life span A and measuring S and I in units of ϱ. The new dimensionless variables $s = S/\varrho$ and $i = I/\varrho$ give the fraction of a cohort which is susceptible and infectious respectively.

The rescaled equations become

$$\frac{\partial s}{\partial a}+\frac{\partial s}{\partial t} = -\lambda s - \mu(a)s$$

$$\begin{aligned} \frac{\partial i}{\partial a} + \frac{\partial i}{\partial t} &= \lambda s - \frac{1}{\varepsilon} i - \mu(a) i \\ \lambda(t) &= \frac{b}{\varepsilon} \int_0^\infty i \, d\alpha \\ s(0,t) &= 1 \qquad i(0,t) = 0, \end{aligned} \tag{2}$$

where the two dimensionless parameters are $b = \beta N D$ and $\varepsilon = D/A$ and the function $\mu(a)$ gives the host mortality in rescaled variables.

The ratio between the two time scales ε now appears explicitly in the model. Furthermore the rescaling shows that all the necessary biological information is summarized in b, ε, and $\mu(a)$. Especially the complicated question of the relationship between disease transmission β and population size N is concentrated in the estimate of b.

Our assumptions about the magnitude b, ε and $\mu(a)$ can not be motivated within the model but must come from biological considerations. We will focus on viral infections with short infectious period relative to the host life span; hence we assume that $\varepsilon \ll 1$. The parameter b is closely related to the reproduction ratio R_0 since R_0 gives the number of secondary infections per primary infection in a susceptible population, and b gives the same quantity if we neglect deaths during the infections period. In the next section we provide some more details about this reasoning but the present argument suffices since the assumption that $\varepsilon \ll 1$ allows us to neglect deaths during infection. The magnitude of R_0 can be assessed in several ways. The probability that an individual escapes infection during its entire life is approximately $1/R_0$ [39] and age stratified serological profiles or case notifications yield information about R_0 [4, 5, 8, 20]. In general one finds that R_0 is on the order of 2–20, the latter corresponding to an extremely contagious disease, Table 1. Therefore we chose to assume that $b = O(1)$.

The mortality $\mu(a)$ increases rapidly with age for old individuals, and $\mu(a)$ introduces large numbers that can not be related to ε. In fact these large numbers will not affect our analysis, but to clarify the approximations, we will avoid using $\mu(a)$ and in stead describe host mortality by the distribution of life lengths with density function

$$g(a) = \mu(a) e^{-\int_0^a \mu(\alpha)\, d\alpha}$$

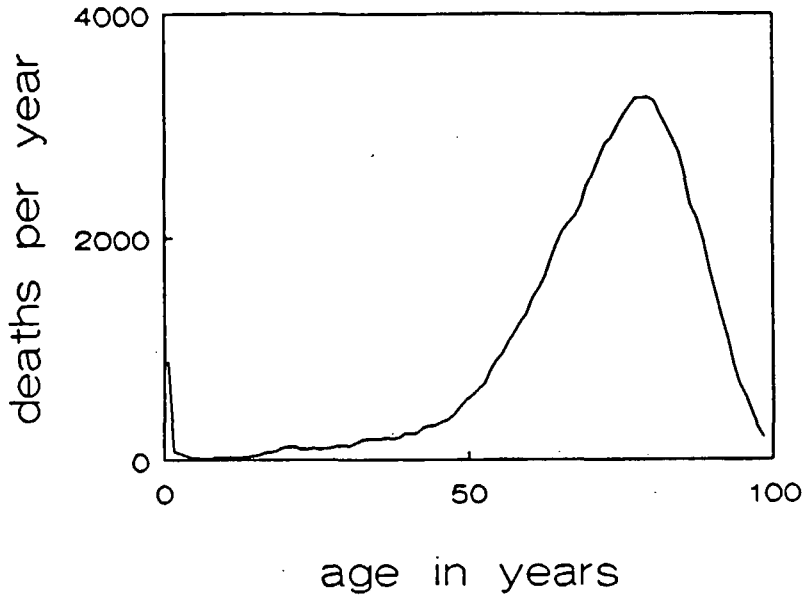

Figure 1. Number of deaths per year for a cohort of 100,000 Danish men, based on the vital statistics for 1984–85. In 1984–85 the expectation of life for Danish men was 71.6 years. Data from [18].

and by the survival function

$$G(a) = e^{-\int_0^a \mu(\alpha)\,d\alpha},$$

giving the probability that a person survives to age a.

Figure 1 shows a typical example of the life length distribution. Due to our rescaling of the age-axis, we know that the mean value of the life lengths is exactly one, $\int_0^\infty a g(a)\,da = 1$. In rescaled variables $g(a)$ is on the order of unity and vanishes for a larger than some $a_0 = O(1)$. In order to allow for convenient examples of $g(a)$ we will not require that $g(a)$ has compact support but assume that $G(a)$ goes to 0 at an exponential rate. In the computations we also will need to know that

$$\int_0^\infty e^{-a/\varepsilon} g(a)\,da \ll \varepsilon^2, \tag{3}$$

corresponding to the requirement that the life length distribution does not have a significant mass concentrated near $a = 0$. For human populations there is a

significant infant mortality, but we will postpone this problem to the discussion and assume that (3) holds.

In conclusion we rescale the SIR model such that the presence of the small quantity ε appears explicitly, while we assume — with some biological justification — that all other parameters are of order unity. The magnitudes of the variables s, i, and λ are not determined, indeed they may vary over time.

2 The equilibrium age distribution

Direct application of asymptotic expansions to (2) does not appear to yield new insights because the magnitude of λ varies. I focus on the analysis of the equilibria and use well known techniques for the local analysis of (2) to derive an implicit equation in λ^*, the force of infection at the endemic equilibrium [12, 25]. I then can show that λ^* is on the order of unity and find a second order expansion of the equation in λ^*.

Any equilibrium age-distribution (s^*, i^*) for (2) can be found by the use of the method of Dietz [20]. The crucial point in Dietz's method is to observe that at equilibrium, the force of infection $\lambda(t)$ is a constant λ^* independent of age. Therefore we can use a two-step process to find λ^*. In the first step, we solve the steady-state equations assuming that λ^* is an (unknown) constant:

$$\begin{aligned} \frac{ds^*}{da} &= -\lambda^* s^* - \mu(a)s^* \\ \frac{di^*}{da} &= \lambda^* s^* - \frac{1}{\varepsilon} i^* - \mu(a)i^* \\ s^*(0) &= 1 \qquad i^*(0) = 0. \end{aligned} \tag{4}$$

It is easy to see that the solution to (4) will remain positive for all $a > 0$, and hence that they correspond to biologically meaningful age distributions [24].

In the second step, we determine λ^* implicitly by requiring that λ^* must satisfy the definition of λ:

$$\begin{aligned} \lambda^* &= \frac{b}{\varepsilon} \int_0^\infty i^* \, da \\ &= \frac{b}{\varepsilon} \int_0^\infty \frac{\lambda^*}{1/\varepsilon - \lambda^*} \left(e^{-\lambda^* a} G(a) - e^{-a/\varepsilon} G(a) \right) da. \end{aligned} \tag{5}$$

The delay kernel corresponding to the distribution of life lengths $g(a)$ does not appear explicitly in the equilibrium condition. To introduce $g(a)$ we integrate (5) by parts and obtain

$$\begin{aligned}\lambda &= \frac{b}{\varepsilon}\frac{\lambda}{1/\varepsilon-\lambda}\left(\frac{1}{\lambda}-\frac{1}{\lambda}\int_0^\infty e^{-\lambda a}g(a)\,da-\frac{1}{1/\varepsilon}+\frac{1}{1/\varepsilon}\int_0^\infty e^{-a/\varepsilon}g(a)\,da\right)\\ &= \frac{b\lambda}{1-\varepsilon\lambda}\left(\frac{1-L_g(\lambda)}{\lambda}-\frac{1-L_g(1/\varepsilon)}{1/\varepsilon}\right),\end{aligned} \tag{6}$$

where for notational convenience we omit the $*$ and let

$$L_g(k)=\int_0^\infty e^{-ka}g(a)\,da$$

denote the Laplace transform of $g(a)$.

It is well known that (6) has at most one positive root for fixed ε, b [22] but for completeness we sketch briefly the argument. The function

$$b(\lambda)=(1-\varepsilon\lambda)\left(\frac{1-L_g(\lambda)}{\lambda}-\frac{1-L_g(1/\varepsilon)}{1/\varepsilon}\right)^{-1}$$

is monotonically increasing for $\lambda > 0$ giving the uniqueness of the positive root. To determine the range of b, first observe that

$$\lim_{\lambda\to 0}\int_0^\infty a\frac{1-e^{-\lambda a}}{\lambda a}g(a)\,da=\int_0^\infty ag(a)\,da=1,$$

where the last equality is due to our rescaling of the age-axis. The range of b is now determined by the limits

$$\lim_{\lambda\to 0}b(\lambda)=b_0=\frac{1}{1-\varepsilon+\varepsilon L_g(1/\varepsilon)}$$

and

$$b\to\infty \quad \text{as } \lambda\to\infty.$$

As already observed, the magnitude of b is closely related to the reproduction ratio R_0. We now provide an asymptotic analysis of the relationship between b and R_0. The lower limit b_0 is the smallest transmission rate that allows the disease to persist. This gives the following condition for the existence of an endemic equilibrium:

$$1 < b/\frac{1}{1-\varepsilon+\varepsilon L_g(1/\varepsilon)}. \tag{7}$$

The right hand side of (7) gives the reproduction ratio R_0 [12, 19, 22]. That is

$$R_0 = b/\frac{1}{1-\varepsilon+\varepsilon L_g(1/\varepsilon)} = b + \varepsilon b + O(\varepsilon^2) \tag{8}$$

Since we know from empirical studies that R_0 typically is on the order of 2–20 for viral diseases, we conclude that for $\varepsilon \ll 1$, the transmission coefficient b is on the order of unity justifying our assumption. We observe by (6) that λ and $1/b$ are of the same magnitude so that $\lambda = O(1)$.

In the next section we will need an expansion of $b(\lambda)$ to second order and we note that for small ε, the equilibrium condition (6) simplifies to

$$\begin{aligned} 1/b &= \frac{1}{1-\varepsilon\lambda}\left(\frac{1-L_g(\lambda)}{\lambda} - \frac{1-L_g(1/\varepsilon)}{1/\varepsilon}\right) \\ &= (1-L_g(\lambda))/\lambda - \varepsilon L_g(\lambda) + O(\varepsilon^2). \end{aligned} \tag{9}$$

To the first order

$$b = \frac{\lambda}{1-L_g(\lambda)}$$

determines the relationship between the disease transmission parameter b and the force of infection at equilibrium λ. Here the effect of the host mortality appears through the Laplace transform L_g of the life length distribution. Since the function e^{-t} is (upward) convex, Jensen's inequality shows that L_g takes its minimum when g is a delta function at $a = 1$, $L_\delta(\lambda) = e^{-\lambda}$ [36, p. 63]. Hence for fixed disease transmission parameter b, the force of infection will be maximal when the host life length is a constant $A(= 1)$. Fixed host life span gives the life length distribution where the fewest deaths occur among susceptibles, and hence where disease incidence peaks. Regarding L_g as a moment generating function [23], we write

$$L_g(\lambda) = e^{-\lambda}\left(1 + \tfrac{1}{2}\mu_2\lambda^2 - \tfrac{1}{3!}\mu_3\lambda^3 + \ldots\right),$$

where μ_j is the j'th moment of $g(a)$ about 1

$$\mu_j = \int_0^\infty (a-1)^j g(a)\, da$$

and $\mu_1 = 0$ by our rescaling of the age-axis. For vanishing higher moments $L_g(\lambda)$ increases with increasing variance μ_2 and hence, for fixed b, the force of

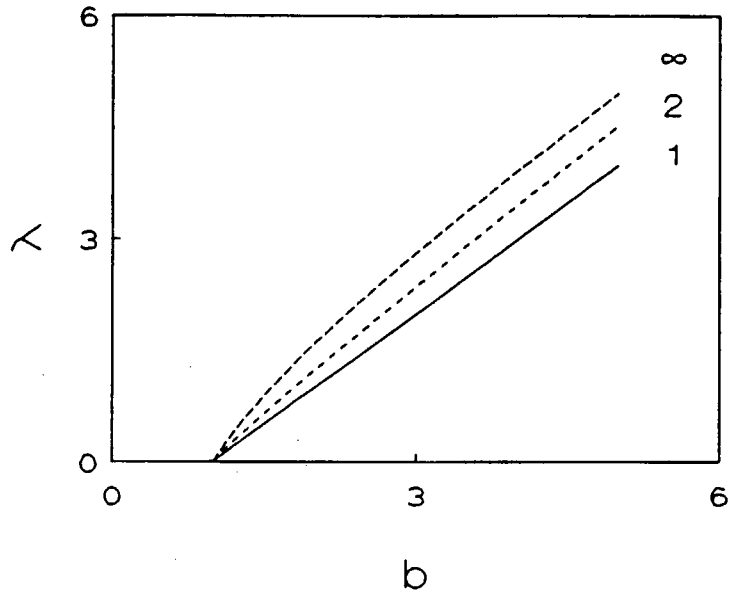

Figure 2. Force of infection λ as a function of the transmission coefficient b in dimensionless units, see text. The curves 1, 2 and ∞ correspond to the life length distributions $g_1(a) = \epsilon^{-a}$, $g_2(a) = 4a\epsilon^{-2a}$, and $g_\infty(a)$, a delta function at 1. For fixed b, $g_\infty(a)$ gives the maximal force of infection.

infection λ decreases with increasing variance. Figure 2 shows $b(\lambda)$ for some life length distributions $g(a)$.

We conclude that at equilibrium, the value of λ is on the order $1/R_0 = O(1)$ and that (9) provides an approximate implicit equation in λ. Often we will regard λ as the basic model parameter and use (9) to determine b. Since there is a one-to-one relationship between b and λ this is formally possible, but it may not be a good parametrization from a biological view point.

3 Stability equation for the endemic equilibrium

Near the endemic equilibrium, (2) contains rates that differ by 3–4 orders of magnitude. Hence, near the equilibrium the proportion of infectious $i(a,t)$ will track closely the proportion of susceptibles $s(a,t)$, i.e. $i(a,t) \approx \lambda(t)s(a,t)\varepsilon$ [3, 33]. Since the magnitude of $\lambda(t)$ is known only near the endemic equilibrium, we cannot use this approach to solve (2) in general. However, the fact that $\varepsilon \ll 1$ facilitates the local analysis of the endemic equilibrium. The local stability analysis leading to the stability equation for the endemic equilibrium is well known and I only sketch the arguments, for details see [12] or others. For $\varepsilon \ll 1$ the stability equation simplifies and I describe the magnitude of the dominant roots and expand for these roots the equation to leading order.

The asymptotic stability of an equilibrium of (2) can be determined by linearizing the equations near the equilibrium and examining separable perturbations of the form

$$\begin{aligned} \hat{\mathbf{s}}(a,t) &= \mathbf{s}(a)e^{pt} \\ \hat{\mathbf{i}}(a,t) &= \mathbf{i}(a)e^{pt} \\ \hat{\theta}(t) &= \theta e^{pt}, \end{aligned}$$

where $\hat{\mathbf{s}}$ and $\hat{\mathbf{i}}$ denote displacements away from the equilibrium values of s and i respectively, while $\hat{\theta}$ is the displacement of λ. The age distributions of the perturbations off the endemic equilibrium must to the first order follow the equations

$$\begin{aligned} \frac{d\mathbf{s}}{da} &= -\lambda^*\mathbf{s} - \theta s^* - p\mathbf{s} - \mu(a)\mathbf{s} \\ \frac{d\mathbf{i}}{da} &= \lambda^*\mathbf{s} + \theta s^* - (p + 1/\varepsilon)\mathbf{i} - \mu(a)\mathbf{i} \\ \mathbf{s}(0) &= \mathbf{i}(0) = 0. \end{aligned}$$

In order for the perturbation to be consistent with the definition of $\hat{\theta}$, we in addition require.

$$\theta = \frac{b}{\varepsilon}\int_0^\infty \mathbf{i}(\alpha)\, d\alpha. \tag{10}$$

This yields the stability equation in the eigenvalue, p,

$$\frac{\varepsilon}{b} = \frac{p}{\lambda(p + 1/\varepsilon)(p + \lambda)} + \frac{\lambda L_g(\lambda + p)}{(\lambda - 1/\varepsilon)p(p + \lambda)}$$

$$-\frac{(p-\lambda)L_g(\lambda)}{\lambda p(p-\lambda+1/\varepsilon)} - \frac{L_g(p+1/\varepsilon)}{\varepsilon(\lambda-1/\varepsilon)(p+1/\varepsilon)(p-\lambda+1/\varepsilon)} \quad (11)$$

where we have used integration by parts to introduce $g(a)$ and its Laplace transform $L_g(k)$ as in the previous section.

The integration of (10) by parts requires that

$$G(a)e^{-(p+\lambda)a} \to 0 \quad \text{for} \quad a \to \infty. \quad (12)$$

If the survival function $G(a)$ is not identically zero for large a, $a > a_0$ (has compact support) there may exist roots of (11) that are not roots of (10) and we restrict our attention to roots of (11) that satisfy (12) ensuring that the roots solve the original stability equation (10).

Equation (11) can not be solved analytically, but the fact that $\varepsilon \ll 1$ allows us to find approximate solutions for the roots near the imaginary axis. These roots give the dominant eigenvalues and hence are the ones that determine the stability of the model.

The term involving $L_g(p+1/\varepsilon)$ can be important only when either $L_g(p+1/\varepsilon)$ is large or when $p \approx -1/\varepsilon$, $\lambda - 1/\varepsilon$. In both cases the real part of p is much smaller than -1, so the term is not relevant for the stability of (2).

To simplify (11) first multiply through by $p(p+\lambda)/\varepsilon$ and omit the order $L_g(1/\varepsilon)$ term:

$$0 = -\frac{p(p+\lambda)}{b} + \frac{p^2}{\lambda(1+\varepsilon p)} - \frac{\lambda L_g(\lambda+p)}{1-\varepsilon\lambda} - \frac{(p^2-\lambda^2)L_g(\lambda)}{\lambda(1+\varepsilon p-\varepsilon\lambda)}.$$

Multiplying through by $p(p+\lambda)$ introduces extraneous roots in (13) at $p = 0, -\lambda$, which do not correspond to suitable perturbations. The equation contains terms that are of different magnitude since by (8) and (9), b and λ are of order 1 while $\varepsilon \ll 1$. The order of p is determined by the method of undetermined gauges, i.e. by trying roots of the form $|p| \approx k\varepsilon^q$ [34, p. 33].

One sees that the p^2/b is unbalanced if $q < -1$. For $q = -1$ the terms of order p^2 and $p^2/(\varepsilon p+1)$ must cancel each other but by (9) this is only possible when $\varepsilon p + 1 = 1 + O(\varepsilon)$. We conclude that $q > -1$ and expand in powers of ε:

$$0 = -\frac{p(p+\lambda)}{b} + \frac{p^2}{\lambda}(1 - \varepsilon p + \varepsilon^2 p^2 + \ldots)$$

$$-\lambda L_g(\lambda+p)(1+\varepsilon\lambda+\varepsilon^2\lambda^2+\ldots)$$
$$-\frac{(p^2-\lambda^2)L_g(\lambda)}{\lambda}(1-\varepsilon(p-\lambda)+\varepsilon^2(p-\lambda)^2+\ldots).$$

Using (9) to remove the p^2 terms and to simplify the εp^3 term, we get

$$\begin{aligned} 0 \;=\; & [-p\lambda/b+\lambda(L_g(\lambda)-L_g(p+\lambda))] \\ & +\varepsilon[-p^3/b-\lambda^2 L_g(\lambda+p)-\lambda(p-\lambda)L_g(\lambda) \\ & +\varepsilon^2[p^4/b]+O(\varepsilon^2 p^3)+O(\varepsilon^3). \end{aligned} \tag{13}$$

By trying roots of the form $|p| \approx k\varepsilon^q$, $q > -1$ one sees that there are only two possible types of other roots for (13) with real part near the imaginary axis, $|p| \approx k + O(\varepsilon)$ and $|p| \approx k\varepsilon^{-1/2} + O(1)$. Notice that the latter type of roots can not be found by taking the limit as $\varepsilon \to 0$ in (13) indicating a singularity in the characteristic equation.

For $|p| \approx k$, the equation becomes

$$-p(1-L_g(\lambda))+\lambda L_g(\lambda)-\lambda L_g(\lambda+p)+O(\varepsilon)=0. \tag{14}$$

For $|p| \approx k\varepsilon^{-1/2}$, the equation is easily solved and we find

$$p = \pm i\sqrt{\lambda/\varepsilon}+O(1). \tag{15}$$

In the last case we need second order terms to determine the stability properties of the roots. We let $p = x + i(y+\sqrt{\lambda/\varepsilon}) + O(\varepsilon^{1/2})$ and find after some tedious calculations and use of (9) that the stability equation reduces to

$$2(1-L_g(\lambda))(x+iy)+\lambda-\lambda L_g(\lambda+p)+O(\varepsilon^{1/2})=0. \tag{16}$$

Since roots with large (positive) real part are excluded, the dominant roots lie near the imaginary axis. For the two types of roots near the axis, we have obtained characteristic equations similar to stability equations for delay differential equations. In the next section we shall analyze these equations and show that all roots have negative real part.

4 The eigenvalues

We shall now analyze the location of the dominant roots of (14) and (16).

First we focus on (14) and the roots $p = x + \mathrm{i}\, y$ near the origin. Using our definition of $L_g(\lambda)$ and retaining only terms of leading order, the left hand side of (14) becomes

$$\begin{aligned} & M_r(p) + \mathrm{i}\, M_i(p) = \\ & \quad -x \int_0^\infty (1 - e^{-\lambda a}) g(a)\, da + \lambda \int_0^\infty (e^{-\lambda a} - \cos ya\, e^{-(\lambda+x)a}) g(a)\, da \\ & \quad -\mathrm{i}\, y \int_0^\infty (1 - e^{-\lambda a}) g(a)\, da + \mathrm{i}\, \lambda \int_0^\infty \sin ya\, e^{-(\lambda+x)a} g(a)\, da. \end{aligned} \tag{17}$$

Since we introduced extraneous roots at $p = 0, -\lambda$, we find $M(0) = M(-\lambda) = 0$, but these roots are not roots of the stability equation (11).

We will now show: *The equation*

$$M_r(p) + \mathrm{i}\, M_i(p) = 0 \tag{18}$$

has only two real roots $p = 0, -\lambda$ and all complex roots have real part less than $-\lambda$.

If $p = x \neq 0, -\lambda$ is real we observe that

$$M_r(p) = \lambda x \int_0^\infty \left(\frac{e^{-\lambda a} - 1}{\lambda a} - \frac{e^{-(\lambda+x)a} - e^{-\lambda a}}{xa} \right) a g(a)\, da$$

For fixed a the left term of the integrand is the difference between the slopes of two secants of e^{-t}. The first secant goes from 0 to λa and the second secant from λa to $(\lambda + x)a$. The relative positions of the values 0, λa, and $(\lambda + x)a$ will not change as $a > 0$ varies and hence the difference between the slopes will not change sign because e^{-t} is (upward) convex. We conclude that the integrand will not change sign and therefore that $M_r(p) \neq 0$ for $p \neq 0, -\lambda$ real.

All non-zero roots of (18) have negative real part. To see this first assume that $p = x + \mathrm{i}\, y$ and $x \geq 0$, $y > 0$. After an elementary rearrangement, the imaginary part of (17) to first order satisfies

$$-M_i(p)/\lambda \;\; = \;\; \int_0^\infty ya \frac{1 - e^{-\lambda a}}{\lambda a} g(a)\, da - \int_0^\infty \sin ya\, e^{-(\lambda+x)a} g(a)\, da$$

$$\geq \int_0^\infty ya e^{-\lambda a} g(a)\, da - \int_0^\infty \sin ya\, e^{-(\lambda+x)a} g(a)\, da$$
$$\geq \int_0^\infty (ya - \sin ya) e^{-(\lambda+x)a} g(a)\, da,$$

which is strictly positive because $\theta > \sin\theta$ for positive θ. Hence complex roots with non-negative real part can not occur.

For complex roots with negative real part $x < 0$, observe that

$$\begin{aligned} M_r(p) &= x\lambda \int_0^\infty \left(\frac{e^{-\lambda a} - 1}{\lambda a} - \frac{e^{-(\lambda+x)a} \cos ya - e^{-\lambda a}}{xa} \right) a g(a)\, da \\ &\geq x\lambda \int_0^\infty \left(\frac{e^{-\lambda a} - 1}{\lambda a} - \frac{e^{-(\lambda+x)a} - e^{-\lambda a}}{xa} \right) a g(a)\, da \end{aligned}$$

Regarding the left hand term of the integrand as the difference between the slopes of two secants of e^{-t}, one sees that the expression is positive if $0 < \lambda + x < \lambda$ so that equation (18) can have roots only if $x < -\lambda$. This concludes our observations on the roots of (18).

For roots $p = x + \mathrm{i}(y + \sqrt{\lambda/\varepsilon})$, the leading term in the equation is

$$\begin{aligned} &N_r(p) + N_i(p) = \\ &2x \int_0^\infty (1 - \epsilon^{-\lambda a})\, g(a)\, da + \lambda \int_0^\infty \left(1 - e^{-(\lambda+x)a} \cos(y + \sqrt{\lambda/\varepsilon})a\right) g(a)\, da \\ &+\mathrm{i}\, 2y \int_0^\infty (1 - \epsilon^{-\lambda a})\, g(a)\, da + \mathrm{i}\, \lambda \int_0^\infty e^{-(\lambda+x)a} \sin(y + \sqrt{\lambda/\varepsilon})a\, g(a)\, da \end{aligned}$$

We first show: *All roots of*

$$N_r(p) + \mathrm{i}\, N_i(p) = 0 \tag{19}$$

have real part less than x_0 where $x_0 < 0$ is the root of

$$2\frac{1 - L_g(\lambda)}{\lambda} x + 1 = L_g(\lambda + x). \tag{20}$$

The real part $N_r(p)$ can be bounded from below by

$$N_r(p)/\lambda \geq 2\frac{1 - L_g(\lambda)}{\lambda} x + 1 - L_g(\lambda + x) \tag{21}$$

so (19) can have roots only if the right hand side of (21) is negative. The function

$$l(x) = 2\frac{1 - L_g(\lambda)}{\lambda}x + 1$$

is increasing with $l(0) = 1$ while the function

$$f(x) = L_g(\lambda + x) = \int_0^\infty e^{-(\lambda+x)a} g(a)\, da$$

is decreasing with $f(0) = L_g(\lambda) < 1$. The two functions will coincide at some $x_0 < 0$ and the right hand side of (21) is positive for $x > x_0$. This gives an upper bound for the real part of the roots of (19).

We can not provide a better general estimate of the roots p of (19) but if $g(a)$ is a continuous function with compact support (or with a sufficiently fast exponential decay as $a \to \infty$.), then we have

$$|L_g(\lambda + p)| = O(\varepsilon^{1/2}),$$

so that the real part of the roots of (19) are located near the real number

$$p = \frac{1}{2}\frac{-\lambda}{1 - L_g(\lambda)} = -\tfrac{1}{2}b.$$

We conclude that for any distribution of life lengths, the characteristic roots are negative and bounded away from the imaginary axis, showing that the endemic equilibrium of (1) is locally asymptotically stable, and that the stability equation has roots with large imaginary part near the imaginary axis. For $L_g(\lambda) < \frac{1}{2}$ the roots $p = -\frac{1}{2}b \pm \mathrm{i}\sqrt{\lambda/\varepsilon} + \mathrm{i}\,O(1)$ dominate; is is not clear if in general these roots are always the dominant ones.

5 Examples

To obtain further information about the position of the two kinds of roots, we examine in more detail the stability equations (14) and (16) for three types of delay kernel. Cushing [17] and MacDonald [32] note that the stability equation for distributed delay models is particularly simple if the delay kernel is taken to be either a gamma distribution of integer order or a discrete delay of fixed length. As the order tends to infinity the width of the gamma distribution

narrows and the gamma distributed delay tends to the discrete delay. The gamma distribution of 0 order correspond to a constant mortality, and we treat this case separately since we here can obtain the exact spectrum. Thus our family of distributions provide a range of life length distributions spanning form constant mortality to constant life length.

Constant mortality

For a constant mortality $\mu = 1$, the SIR-model is age independent and the characteristic roots are easily determined by linear analysis [20, 26]. In our notation we get

$$p = -\frac{\lambda+1}{2} \pm \sqrt{(\lambda-1)^2/2 - \lambda/\varepsilon} = -\frac{1}{2}b \pm \sqrt{(\lambda-1)^2/2 - \lambda/\varepsilon}.$$

Since we still have $\lambda = O(1)$, the roots have large imaginary part of the form $\sqrt{\lambda/\varepsilon} + O(1)$ and negative real part $-\frac{1}{2}b$ as expected. The approximate result may also be found by setting $n = 1$ in the next section.

Gamma distributed delay

Since the age-axis is rescaled to ensure that the mean of the distribution is unity, we chose gamma distributions with mean 1. For a gamma distributed delay of integer order $n-1$, the form parameter must equal n^{-1} and the density function becomes

$$g_n(a) = \frac{n}{(n-1)!}(an)^{n-1}e^{-an}. \qquad n \geq 1 \tag{22}$$

We let $G_n(a)$ denote the survival function corresponding to $g_n(a)$. A simple computation shows that the Laplace transform is

$$L_g(k) = (1 + k/n)^{-n}.$$

To bring the stability equation (10) on the form (11), we performed an integration by parts which was only possible if $G_n(a)e^{-(\lambda+p)a} \to 0$ for $a \to \infty$. Hence our analysis applies only to characteristic roots p that satisfy $\mathrm{Re}\, p > -(\lambda+n)$.

The stability equation (14) for roots p near the origin is

$$-p\left(1-(1+\lambda/n)^{-n}\right)+\lambda(1+\lambda/n)^{-n}-\lambda\,(1+(\lambda+p)/n)^{-n}=0, \qquad (23)$$

where we have retained only terms of leading order. As already observed we have introduced for algebraic simplicity roots $p=0,-\lambda$ that are not roots in the characteristic equation. By a modification of the method of Blythe et al. [10], I prove in appendix A that all non-zero roots expect the one at $-\lambda$ have real part less than x_1 where x_1 is determined by

$$x_1=-(n+\lambda) \qquad \text{if } n\le 4;$$

and by

$$\left(1+\frac{x_1}{n+\lambda}\right)^{-2n}\cos^{2n}\frac{2\pi}{n}\ge(1-cx_1)^2+c^2(n+\lambda+x_1)^2\tan^2\frac{2\pi}{n} \qquad \text{if } n>4, \qquad (24)$$

where $c=[(1+\lambda/n)^n-1]/\lambda$.

The real part x_2 of the order $\varepsilon^{-1/2}$-roots is determined by

$$2(x_2+\mathrm{i}y_2)\frac{1-(1+\lambda/n)^{-n}}{\lambda}+1-\left(1+\frac{x_2+\mathrm{i}y_2+\mathrm{i}\sqrt{\lambda/\varepsilon}+\lambda}{n}\right)^{-n}+O(\varepsilon^{1/2})=0. \qquad (25)$$

Omitting all terms of order $\varepsilon^{1/2}$ or less, we get

$$x_2=-\frac{1}{2}\frac{\lambda}{1-(1+\lambda/n)^{-n}}=-\frac{1}{2}b. \qquad (26)$$

Elementary algebra shows that $-(\lambda+1)/2<x_2<-\lambda/2$. Since in general the order 1 roots have real part less than $-\lambda$, this shows that the order $\varepsilon^{-1/2}$-roots dominate for $\lambda>1$. For $\lambda<1$, one can show that the upper bound x_1 is a decreasing function of λ with $\lim_{\lambda\to 0}x_1(\lambda)<-1$. Hence the order $\varepsilon^{-1/2}$-roots dominate in this case as well.

Discrete delay

When all individuals have exactly the same life length, $g(a)$ is a delta function at 1. In this case the approximate stability equations (14) and (16) are valid

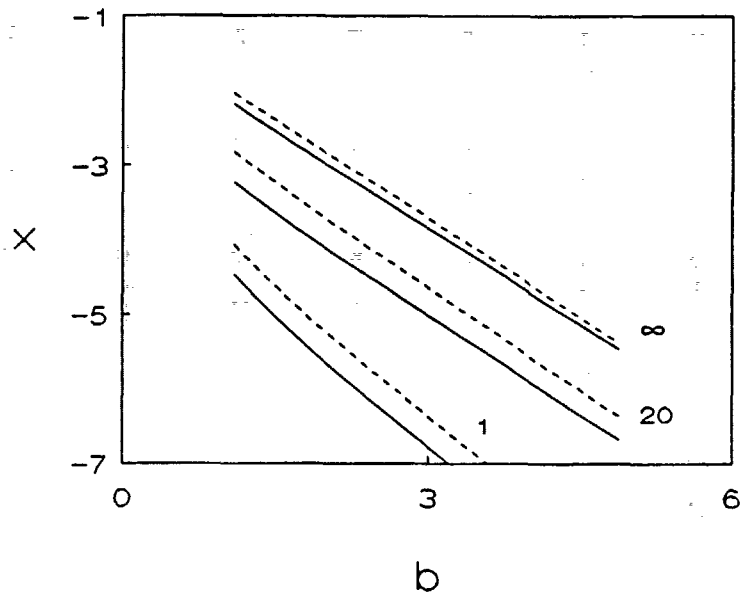

Figure 3. The real part x of roots of type $p = O(1)$ for the stability equation (11) when life lengths follow a gamma distribution of order $n-1$ and when life length is fixed ($n = \infty$). The solid lines show the dominant root found by numerical solution of (23) and the broken lines indicates the upper bound (24). In addition to these roots the model has roots with real part $-\frac{1}{2}b + O(\varepsilon^{-1/2})$ and large imaginary part.

for all roots near the imaginary axis since the survival function $G(a)$ vanishes for $a > 1$. We find,

$$L_g(k) = e^{-k},$$

so that the stability equation for the order 1 roots takes the form

$$-p(1 - e^{-\lambda}) + \lambda e^{-\lambda}(1 - e^{-p}) + O(\varepsilon) = 0 \tag{27}$$

In appendix A we prove that all roots of (27), except the ones at 0 and at $-\lambda$ have real part less than x_1 where x_1 is given implicitly by

$$e^{-2x_1} = 4\pi^2 c^2 + (1 - cx_1)^2, \tag{28}$$

and $c = (e^{\lambda} - 1)/\lambda > 0$.

For the roots near $i\sqrt{\lambda/\varepsilon}$, the general estimate (20) provides an upper bound x_0. A heuristic argument, in Appendix B, suggests that as b varies,

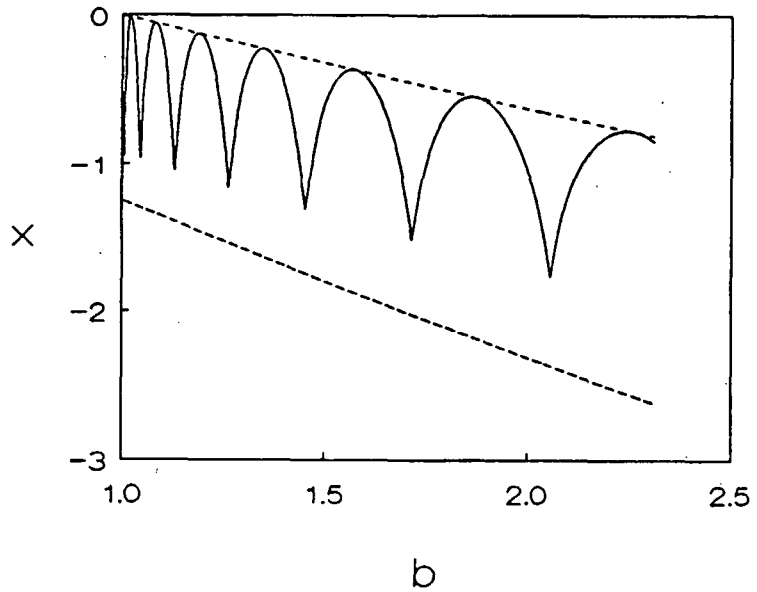

Figure 4. The real part x of roots of type $p = \pm i\sqrt{\lambda/\varepsilon}$ for the stability equation(11) when life length is fixed and and the ratio between the two time scales is $\varepsilon = 10^{-3}$. Notice that x is sensitive to small changes in the transmission coefficient b. The two broken lines give the upper bound x_0 (20) and a heuristically derived lower bound x_m, see Appendix B. The roots are found numerically by a modification of Laguer's method [35, p. 263].

the real part of dominant eigenvalues will fluctuate rapidly within an interval $[x_m; x_0]$. Numerical solutions of (27) support this claim (Figure 4).

Numerical solutions show that the order $\varepsilon^{-1/2}$ roots are the dominant ones, and that the heuristically derived lower bound for the real part of the order $\varepsilon^{-1/2}$-roots x_m is always larger than x_1, the upper limit for the real part of the order 1 roots. Therefore the stability is characterized by the weakly damped oscillations oscillations of intermediate period associated with the roots of type (15).

The results of this section are summarized in Figure 3 showing the largest real part of order ε^0 roots, and Figure 4 showing the real part of the order $\varepsilon^{-1/2}$ in the model with fixed life span.

As n increases the gamma distribution $g_n(a)$ narrows around $a = 1$ and in the limit we obtain the discrete delay. The equation for the ε^0-order roots reflects this observation in that the equation for the gamma distribution (25) will tend to the equation with discrete delay (27) as n goes to infinity. However our estimate of the real part of the $\varepsilon^{-1/2}$ roots for $g_n(a)$ (26) do not behave in this way since in the limit we can not neglect the $(1 + \varepsilon/n)^{-n/2}$-term and for the discrete delay the leading term in the $i\sqrt{\lambda/\varepsilon}$-roots will depend critically on ε.

6 Discussion

The presence of an endemic infectious disease is due to a balance between the introduction of new susceptibles and the loss of susceptibles through infection and subsequent recovery or death. For a disease that confers permanent immunity, new susceptibles appear only through host births, and the time scale of this process is closely linked to the host life span [44]. Susceptibles become infected through contact with infectious individuals so the course of the disease within the individual host determines the disease transmission. The maintenance of an immunizing disease thus depends on the interaction between two biologically distinct phenomena. The key observation in this paper is that for many infectious diseases, host renewal and individual infection take place on time scales that differ by 3–4 orders of magnitude.

After a rescaling of time to units of the average host life span and 'non-dimensionalization' of the variables, the ratio ε between the two time scales appears explicitly in the age-dependent SIR-model. The model contains only two additional dimensionless parameters, b that describes disease transmission and $g(a)$ describing the distribution of host life-lengths in the rescaled variable. From biological considerations we chose to assume that $b = O(1)$ and $g(a) = O(1)$. In the rescaled system the well known threshold condition for the existence of an endemic equilibrium in the SIR-model becomes $b > 1 + O(\varepsilon)$, and we focus on this situation and on how the mortality structure affects the equilibrium for fixed mean host life span and mean duration of infection.

An asymptotic expansion of the equilibrium condition yield a simple, implicit relationship between b, $g(a)$ and λ, the force of infection at equilibrium.

For fixed b the force of infection λ takes its maximum when the distribution of host life-lengths $g(a)$ correspond to a fixed life span A, and — for vanishing higher moments of g — λ decreases with increasing variance around A. Narrowing the life length distribution around the mean reduces the number of deaths in the younger age-classes and since younger individuals are more likely to be susceptible, a narrow distribution gives a higher incidence of the disease.

The stability equation for the endemic equilibrium takes a form similar to that of delay differential equations with delay kernel $g(a)$. In epidemiology delay models have been studied in connection with variable infectivity or period of infectivity [11, 15, 27] and age-dependent mortality [41]. For the age-dependent model with the present limit procedure, the structure of the characteristic equation is considerably simpler, allowing us to describe in some detail the dominant part of the spectrum of the endemic equilibrium. By asymptotic expansion of the stability equation we find that all roots have negative real part, bounded away from the imaginary axis, and that the roots near the imaginary axis must be complex. Thus the endemic equilibrium is always stable when it exists. Using gauges of the form $|p| = k\varepsilon^q$, we find that the stability equation is singular at $\varepsilon = 0$ and that two types of roots near the axis may occur: 1) roots located near the origin and 2) roots near $\pm i\sqrt{\lambda/\varepsilon}$.

For continuously distributed life lengths that fall off sufficiently fast as age goes to infinity, the roots near $\pm i\sqrt{\lambda/\varepsilon}$ have real part $x = -\frac{1}{2}b + O(\varepsilon^{-1/2})$, the same as in the model with constant mortality. In this case the damping time as measured by $\frac{1}{2}b$ is independent of the details of the mortality structure while the period determined from the imaginary part $\sqrt{\lambda/\varepsilon}$ will change with the life length distribution $g(a)$. If $g(a)$ has peaks of width $O(\varepsilon^{-1/2})$, the leading term in real part of the order $\varepsilon^{-1/2}$-roots fluctuates with small changes in parameter values. For example, for a population with fixed life length, I give a rigorous upper bound for x and a heuristically derived lower bound. Within those bounds, x appears to be sensitive to variation in the transmission factor b.

If life lengths follow a gamma distribution, the stability condition may be expressed in algebraic terms, allowing for an explicit bound on the roots and numerical solution of the spectrum. Here the roots with large imaginary part always dominate. The waiting time for the completion of n consecutive exponential decays follow a gamma distribution of order n, so the analysis applies

to such models. Tudor [42] and Schenzle [37, 38] proposed a discretization of the age-structured SIR-model obtained by dividing the host population into n (age) groups with a constant transfer rate from one group to the next and variable mortality and disease transmission among groups. Thus model (2) with a gamma distributed delay correspond to their models in the case where all transmission rates are identical and all mortality occur in the highest age class. Our computation confirms Tudor and Schenzle's numerical findings that their models are always stable.

For any life length distribution $g(a)$, the order $\varepsilon^{-1/2}$-roots are dominant for sufficiently large λ and appear to always dominate. The analysis thus confirms the general observation that age-structured models in epidemiology exhibit slowly damped oscillations with period $T = 2\pi\sqrt{\varepsilon/\lambda}$ or in dimensional units $T_u = 2\pi\sqrt{DA/\lambda}$ where A and D are the period of infectiousness and the host life span respectively. Since we find that $\lambda = O(1)$, the period is on the order of the geometric average of the two times scales involved [3].

Throughout the paper we have assumed that the distribution of deaths has no significant mass concentrated at age 0, c.f. condition (3). A close examination of Figure 1 shows that even for industrialized countries, infant mortality can not be neglected and hence our condition (3) on the distribution of life-lengths $g(a)$ may not be biologically reasonable. In practice the effect of infant mortality on disease transmission dynamics may be small: Infants are temporarily protected against many infectious diseases by maternal antibodies and much of the mortality occurs in premature infants that are isolated in hospitals' intensive care units. The infant mortality will affect the reproduction of hosts, but since we have not accounted in detail for the host population size and regulation, childhood mortality simply may be compensated by a slight increase in the birth rate ϱ. For developing countries, however, condition (3) may be unrealistic and our results may not hold.

In addition to the demographic effect on disease transmission dynamics, epidemiological evidence show that disease transmission depends on the age of both infector and infectee [4, 37]. A change of variables from the age-distribution of each of the epidemic classes to age-specific frequencies of the epidemic classes shows that the survival function $\exp(\int_o^a -\mu(\alpha)\,d\alpha)$ plays the same role in the dynamics as age-dependent infectivity [13, 29, 41]. Therefore

it is straight forward to generalize our results allowing for disease transmission to depend on the age a of the infectors. The delay kernel in this case becomes

$$h(a) = (\mu(a)\beta(a) - \beta'(a))\, e^{\int_0^a -\mu(\alpha)\, d\alpha},$$

where $\beta(a)$ is the age-dependent transmission coefficient in rescaled variables. With restrictions on the range and variation of $h(a)$ similar to (3) one can show that the dominant part of the spectrum has the same structure as in the case $\beta(a) = b$. We have no biological reason to assume that $h(a) > 0$ but the proof may be modified to show that the roots near the origin remain in the negative half plane though the upper bound on the real part is weaker. If $h(a)$ contains no point masses, the roots near $i\sqrt{\lambda/\varepsilon}$ will remain in the negative half plane since the real part is independent of h. The analysis of age-dependent disease susceptibility is considerably more complicated. A partial analysis shows that the roots with large imaginary part can cross the imaginary axis when disease susceptibility decreases with age [7].

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Appendix

A The 0 order equation.

In case of gamma distributed life-lengths the characteristic equation for the 0 order roots p simplifies to

$$1 - \gamma z = (1 + z)^{-n} \tag{29}$$

where $\gamma = (n + \lambda)[(1 + \lambda/n)^n - 1]/\lambda$ and the eigenvalues are rescaled by $z = p/(n+\lambda)$. In the main text we introduced extraneous roots in the stability equation at $p = 0$ and $p = -\lambda$ corresponding to $z = 0, -\lambda/(n + \lambda)$. All other roots are complex, come in conjugate pairs, and must have $\mathrm{Re}\, p < -\lambda$ Furthermore only roots with real part $\mathrm{Re}\, p > -(n + \lambda)$ will correspond to roots for the stability equation. We focus on such roots $z = \xi + \mathrm{i}\eta$ with $-\lambda/(n + \lambda) > \xi > -1$ and $\eta > 0$.

Following [10], we reparametrize the problem in terms of the real part ξ of z and the principal argument θ of $1 + z$, i.e.

$$\tan\theta = \frac{\eta}{1+\xi} \qquad 0 \le \theta < \frac{\pi}{2}.$$

as η varies from 0 to ∞, θ will run from 0 to $\pi/2$.

For fixed ξ, $-\lambda/(\lambda + n) > \xi > -1$ we now define two functions

$$L_\theta = 1 - \gamma z = 1 - \gamma\xi - \mathrm{i}\,\gamma(1 + \xi)\tan\theta$$

and

$$S_\theta = (1 + z)^{-n} = (1 + \xi)^{-n}\cos^n\theta e^{-\mathrm{i}\,n\theta}.$$

Solutions to equation (29) will correspond to values of θ where the two functions take the same (complex) value. As θ varies from 0 to $\pi/2$, L_θ makes a half line in the IV-quadrant with base point $z = 1 - \gamma\xi > 0$ while the value of S_θ describes a shrinking spiral turning clockwise from $z = (1 + \xi)^{-n}$ to $z = 0$, see Figure 5.

I first prove that if θ satisfies the inequality

$$\tan n\theta > n\tan\theta > 0 \tag{30}$$

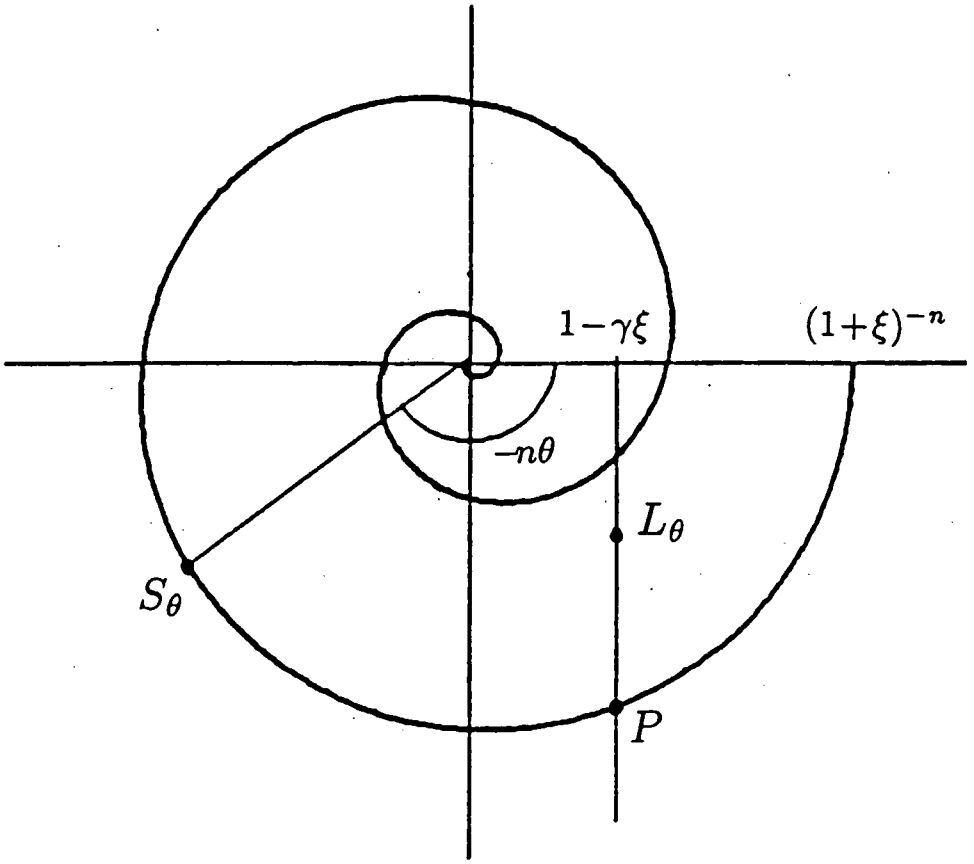

Figure 5. Position of the points $L_\theta = 1 - \gamma z$ and $S_\theta = (1+z)^{-n}$ for fixed real part $\operatorname{Re} z = \xi$ and varying argument θ of $1+z$. At the point P where the spiral S_θ crosses the line L_θ for the first time, $\operatorname{Im} L_\theta < \operatorname{Im} S_\theta$.

then L_θ lies above S_θ for the angle θ where S_θ crosses the $1 - \gamma z$-line, i.e. I prove that if (30) holds then

$$\operatorname{Re} L_\theta = \operatorname{Re} S_\theta \Rightarrow \operatorname{Im} L_\theta > \operatorname{Im} S_\theta.$$

To see this first observe that by the convexity of the function $(1+t)^n$, the constant γ satisfies the inequality

$$(n+\lambda)\left(1+\frac{\lambda}{n}\right)^n > \gamma > n+\lambda.$$

Since $\xi < -\lambda/(n+\lambda)$ we find that when $\operatorname{Re} L_\theta = \operatorname{Re} S_\theta = 1-\gamma\xi$, the imaginary part gives

$$\operatorname{Im} L_\theta \;=\; -\gamma\eta =$$

$$-\gamma(1+\xi)\tan\theta > -\left(1+\frac{\lambda}{n}\right)^n n\tan\theta$$

and

$$\begin{aligned}\operatorname{Im} S_\theta &= -(1-\gamma\xi)\tan n\theta < \\ &\quad -\left(1+\frac{\lambda}{n}\right)^n \tan n\theta. \end{aligned} \tag{31}$$

So if (30) holds, then $\operatorname{Im} L_\theta > \operatorname{Im} S_\theta$. Condition (30) holds for $n\theta < \pi/2$ so since S_θ starts on the right hand side of the origin when $\xi > -1$, S_θ must have turned at least $\pi/2$ before a root can occur. Since the L_θ lies in the IV-quadrant, S_θ must have completed a full turn and $n\theta > 2\pi$.

We conclude that $\theta > 2\pi/n$. By our parametrization we have that $\theta < \pi/2$. For $n \leq 4$, $n\theta < 2\pi$, so in this case roots with $\xi > -1$ are excluded.

For $n > 4$ roots with $\xi > -1$ may exist, and we use the condition $\theta > 2\pi/n$ to obtain an upper bound on ξ by comparing the modulus of L_θ and S_θ. The conditions $\pi/2 > \theta > 2\pi/n$ and $\xi > -1$ gives

$$|S_\theta| = (1+\xi)^{-n}\cos^n\theta \leq (1+\xi)^{-n}\cos^n\frac{2\pi}{n},$$

and

$$|L_\theta|^2 = (1-\gamma\xi)^2 + \gamma^2(1+\xi)^2\tan^2\theta \geq (1-\gamma\xi)^2 + \gamma^2(1+\xi)^2\tan^2\frac{2\pi}{n}.$$

At a root, $|L_\theta|^2$ must equate $|S_\theta|^2$ providing an upper bound on ξ:

$$(1+\xi)^{-2n}\cos^{2n}\frac{2\pi}{n} \geq (1-\gamma\xi)^2 + \gamma^2(1+\xi)^2\tan^2\frac{2\pi}{n}.$$

In the case of a discrete delay, the analysis is quite similar. To leading order the stability equation (27) is

$$1 - cp = e^{-p} = 0, \tag{32}$$

where $c = (e^\lambda - 1)/\lambda > 0$.

Apart from the two extraneous roots $p = 0, -\lambda$ all roots are complex Setting $p = x + iy$, we get

$$1 - cx = e^{-x} \cos y \tag{33}$$
$$cy = e^{-x} \sin y. \tag{34}$$

Focusing on the roots with positive imaginary part, $y > 0$, we observe that since $\sin y < y$, (34) shows that $c < e^{-x}$ or $x < -\log c < 1/c - 1$, so that

$$c < 1 - cx.$$

Rewriting (33)-(34) in terms of modulus and argument gives

$$cy/(1 - cx) = \tan y \tag{35}$$
$$cy^2 + (1 - cx)^2 = e^{-2x}. \tag{36}$$

Simple geometry and (35) now show that $y > \pi/2$. Since $1 - cx > c > 0$, (33) shows that $\cos y > 0$ and hence that $y > 3\pi/2$. Equation (34) shows that also $\sin y > 0$ so that $y > 2\pi$. The modulus equation (36) now yields

$$e^{-2x} = c^2 y^2 + (1 - cx)^2 > 4\pi^2 c^2 + (1 - cx)^2,$$

giving an upper limit for the value of x.

B Second order approximation to order $1/\sqrt{\varepsilon}$-roots

In the case of fixed life length, the stability equation (16) for the roots $p = x + \mathrm{i}\,(y + \omega)$, $\omega = \sqrt{\lambda/\varepsilon}$ becomes

$$2(1 - e^{-\lambda})(x + \mathrm{i}\, y) + \lambda - \lambda e^{-(\lambda+x)a}(\cos(y + \omega) - \mathrm{i} \sin(y + \omega)) = 0$$

which depends explicitly on ε.

With $c = (e^{\lambda} - 1)/\lambda > 0$, we have

$$e^{-x} \cos(y + \omega) = 2xc + e^{\lambda}$$
$$-e^{-x} \sin(y + \omega) = 2yc.$$

It follows that

$$e^{-2x} = (2xc + e^{\lambda})^2 + (2yc)^2. \tag{37}$$

The real part of the order $\varepsilon^{-1/2}$ eigenvalue x is determined by the intersection of the graph for e^{-2x} and the parabola $(2cx + e^{\lambda})^2 + (2cy)^2$. The rightmost intersection of e^{-2x} and the parabola $(2cx+e^{\lambda})^2$ gives our general upper bound x_0 (20) on x.

We now describe the roots as a function of λ by the following heuristic argument. The imaginary, second order correction term, y, is determined by the equation

$$\tan(y + \omega) = -\frac{2c}{2xc + e^{\lambda}} y. \tag{38}$$

I conjecture that (38) has a root between $-\pi/2$ and $\pi/2$. (This is not clear, since (38) depends on x, but I argue that x does not matter in the sense that for any given x, (38) has at least one solution between $-\pi/2$ and $\pi/2$.) The equation will have additional roots with larger magnitude of y, but by (37) they cannot give rise to the dominant order $\varepsilon^{-1/2}$ eigenvalue. Since $\omega = \sqrt{\lambda/\varepsilon}$, ω changes rapidly when λ varies, and we expect that the roots of (38) will vary rapidly between $-\pi/2$ and $\pi/2$. The variation in y will force x to vary between x_0 and x_m, where x_m is the intersection between e^{-2x} and the parabola $(2cx + e^{\lambda})^2 + (c\pi)^2$ (Figure 4).

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Instability in an SIR-model with age-dependent susceptibility

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Abstract: For many common contagious diseases, the infectious period is several orders of magnitude smaller than host life span while the transmission potential R_0 is on the order of unity. If such a disease confers permanent immunity, its persistence depends on biological processes occurring on two different time scales. Therefore many situations of biological interest (e.g. childhood diseases) are covered when applying to the disease models asymptotic expansions in the ratio of the two time scales. In an SIR-model with age-dependent mortality and disease transmission of the proportionate mixing type, a suitable change to dimensionless variables yields a model on the long time scale where all parameters are on the order of unity except $\varepsilon \ll 1$, the ratio between infection period and host life length. The magnitude of the force of infection may change during a transient period and I focus on the stability properties of the endemic equilibrium. For ε small, two types of roots occur near the imaginary axis: Roots near the origin and roots with imaginary part of order $1/\sqrt{\varepsilon}$. The order $1/\sqrt{\varepsilon}$ roots give rise to the oscillations with a period of a few years that are characteristic of SIR-models. If disease susceptibility decreases with age, the eigenvalues with large imaginary part may have positive real part and numerical simulations suggest that a stable limit cycle appears through a Hopf-bifurcation.

Key words: epidemic model, age structure, singular perturbation, Hopf bifurcation.

AMS(MOS): 92A15 (34K20, 35L60)

In this note I describe the structure of the stability equation for the endemic equilibrium of an SIR-model with age-dependent disease transmission and host mortality using the simplifying assumption that the infectious period is small compared to the host life span. The limiting case of short infectious

period is appropriate for many common infections such as childhood diseases or influenza. Anderson and May (1983) used this observation to determine the period – but not the stability – of the oscillations in the model. For this limit, I recently showed that when only mortality depends on age, the endemic equilibrium is always stable with two components in the dominant spectrum, one component near the origin and one singular component with imaginary part on the order of the geometric average of the host life span and the infectious period (Andreasen, 1992). The latter roots concur with the period determined by Anderson and May (1983). Here we relax the assumptions allowing for age-dependent disease transmission following proportionate mixing (Dietz and Schenzle, 1985).

Starting with the works of Hoppensteadt (1974) and Dietz (1975), SIR-type models of infectious disease epidemiology have received much attention during the last 15 years and many basic properties of such models are now well known (Dietz and Schenzle, 1985; Busenberg et al, 1988; Castillo-Chavez et al, 1989; Greenhalgh, 1987, 1988; Busenberg and Hadeler, 1990; Inaba, 1990; Thieme, 1991; Ianelli et al, 1992). The models are well posed; for positive initial data the solutions remain non-negative; and the models exhibit a threshold phenomenon controlling the existence of one (or more) equilibrium solutions with positive disease prevalence. In the situation where such endemic equilibria exist, their local stability may be determined by standard linearization methods yielding a stability equation in the eigenvalues of the associated linear operator. The location of the eigenvalues is known only under quite restrictive assumptions: Inaba (1990) found that the equilibrium is stable for low disease incidence when disease transmission is independent of age while Thieme (1991) showed that high disease levels can lead to instability in the same model.

To focus on the situation where the infectious period is small, we first reparametrize the SIR-model to display explicitly the small parameter. In the subsequent sections we assume that an endemic equilibrium exists and sketch how the implicit expressions for the endemic equilibrium and for the characteristic roots of its linearization are derived in the previously mentioned studies. By applying perturbation methods to these algebraic equations we obtain simple expressions for the leading eigenvalues of the stability equation and show how age-dependent susceptibility may lead to loss of local stability.

1 Dimensional analysis

The basic age-dependent SIR-model that we shall study was formulated by Hoppensteadt (1974) and Dietz (1975) who proposed to describe the dynamics of an epidemic disease without latent period by the following system of differential equations

$$\begin{aligned}
\frac{\partial S}{\partial a} + \frac{\partial S}{\partial t} &= -\Lambda(a,t)S(a,t) - \mu(a)S(a,t) \qquad 0 \le a \le A \\
\frac{\partial I}{\partial a} + \frac{\partial I}{\partial t} &= \Lambda(a,t)S(a,t) - \nu I(a,t) \\
\Lambda(a,t) &= C\int_0^A b(a)\tilde{b}(\alpha)I(\alpha,t)\,d\alpha \\
S(0,t) &= \varrho \\
I(0,t) &= 0.
\end{aligned}$$

Here $S(a,t)$ and $I(a,t)$ denote the age distribution of susceptibles and infectious to time t, while $\Lambda(a,t)$ gives the age-dependent force of infection determined by a weighted average of the infectious at time t. To simplify the discussion we have assumed proportionate mixing so that disease transmission separates into an age-dependent susceptibility $b(a)$, an age-dependent infectivity $\tilde{b}(\alpha)$, and a scaling factor C to be discussed later. All individuals are subject to the same age-dependent mortality $\mu(a)$ and infectious individuals recover and obtain permanent immunity at a rate ν independent of age and time since infection. The boundary conditions indicate that individuals are born susceptible at a fixed rate ϱ and we assume that initial data have been chosen so that the population has already reached demographic equilibrium. Finally we assume that there is a fixed upper limit of host life lengths A.

The model involves two characteristic time scales, the infectious period ν^{-1} and the average host life span

$$L = \int_0^A M(a)\,da,$$

where $M(a) = \exp(-\int_0^a \mu(\alpha)d\alpha)$ denotes the probability of survival to age a. We focus on the long time scale and measure time and age in units of the host life length L, $t = L\bar{t}$ and $a = L\bar{a}$. In the rest of this paper, we refer only to the new dimensionless time and age variables and omit the bar for simplicity.

To further simplify the analysis, we replace S and I by the fractions of the cohort which is susceptible and infectious respectively

$$s(a,t) = \frac{S(a,t)}{\varrho M(a)} \qquad i(a,t) = \frac{I(a,t)}{\varrho M(a)}.$$

In these new coordinates, the model reads

$$\begin{aligned}
\frac{\partial s}{\partial a} + \frac{\partial s}{\partial t} &= -\lambda s \qquad 0 \le a \le A_1 && (1)\\
\frac{\partial i}{\partial a} + \frac{\partial i}{\partial t} &= \lambda s - \frac{1}{\varepsilon} i && (2)\\
\lambda(a,t) &= C\varrho \int_0^{A_1} b(a)\tilde{b}(\alpha) i(\alpha,t) M(\alpha)\, d\alpha && (3)\\
s(0,t) &= 1 && (4)\\
i(0,t) &= 0, && (5)
\end{aligned}$$

where $\varepsilon^{-1} = \nu L$, $A_1 = A/L$ while $b(a)$ and $\tilde{b}(\alpha)$ denote the age-dependent transmission coefficients in the new age-coordinates.

The next step involves biological — not mathematical — considerations in that we wish to assess the magnitude of the parameters. Since we study diseases with short infectious period ν^{-1}, we assume that $\varepsilon = \nu^{-1}/L \ll 1$. For human populations the maximal life span A and the average lifespan L are of the same order of magnitude so that $A_1 = O(1)$ with $M(A_1) = M'(A_1) = 0$ while $M(\alpha) = O(1)$ for $\alpha < A_1$.

The parametrization of the disease transmission coefficient includes an arbitrary scaling factor C so we chose to set $b(a)$ and $\tilde{b}(a) = O(1)$. Since disease transmission varies with age and hence on the slow time scale, we also assume that the first and second derivative of $b(a)$ and $\tilde{b}(a)$ are of order unity. The remaining quantity $C\varrho$ is closely linked to the transmission potential R_0 measuring the number of secondary infections that an 'average' infectious individual will cause in a population comprised of susceptibles only (Diekmann et al, 1990). For proportionate mixing the transmission potential R_0 is known explicitly (Dietz and Schenzle, 1985)

$$R_0 = \int_0^{A_1} C\varrho\tilde{b}(a)M(a) \int_0^a b(\alpha) e^{-\frac{1}{\varepsilon}(a-\alpha)}\, d\alpha\, da.$$

A straight forward computation now shows that

$$R_0 = C\varrho\varepsilon \int_0^{A_1} b(a)\tilde{b}(a)M(a)\,da + C\varrho O(\varepsilon^2).$$

The magnitude of R_0 can be found by independent methods and varies for short lived infections like influenza and childhood diseases from 2 to 20 (Anderson and May, 1991). Therefore we chose to assume that $R_0 = O(1)$ and introduce $c = C\varrho\varepsilon = O(1)$. We can now replace equation (3) by

$$\lambda(a,t) = \frac{c}{\varepsilon}\int_0^{A_1} b(a)k(\alpha)i(\alpha,t)\,d\alpha, \tag{6}$$

where $k(\alpha) = \tilde{b}(\alpha)M(\alpha)$. These observations allow us to assume that in our final model (1,2,6,4, 5) all parameters except ε are of order $O(1)$.

2 The endemic equilibrium and its stability

When the threshold parameter R_0 exceeds unity, the disease-free equilibrium looses its stability and a positive *endemic* equilibrium appears. Since we assume proportionate mixing, the endemic equilibrium is unique (Dietz and Schenzle, 1985). The equilibrium age distribution $s^*(a), i^*(a)$ and its stability equation may be determined by well known methods e.g. (Dietz, 1975; Greenhalgh, 1988) and we only sketch the results; for details see for example (Thieme, 1991). To find an equilibrium one first observes that the force of infection at equilibrium $\lambda^*(a)$ is of the form $\lambda^*(a) = l^* b(a)$ where l^* is a constant yet to be determined. The value of l^* is found by solving the steady state version of (1,2,4,5) for s and i and substituting the solution for i into (6) the definition of $\lambda(a)$ to obtain an equation in l:

$$\lambda(\alpha) = lb(\alpha) \;=\; \frac{c}{\varepsilon}\int_0^{A_1} b(\alpha)k(a)i(a)\,da$$

or

$$\begin{aligned} l &= \frac{c}{\varepsilon}\int_0^{A_1} k(a)\int_0^{a} lb(\alpha)s(\alpha)e^{\frac{1}{\varepsilon}\alpha}\,e^{-\frac{1}{\varepsilon}a}\,d\alpha\,da \\ &= \frac{c}{\varepsilon}\int_0^{A_1}\int_{\alpha}^{A_1} k(a)e^{-\frac{1}{\varepsilon}a}\quad lb(\alpha)s(\alpha)e^{\frac{1}{\varepsilon}\alpha}\,da\,d\alpha \\ &= c\int_0^{A_1} lk(\alpha)b(\alpha)s(\alpha)\,d\alpha + c\varepsilon\int_0^{A_1} lk'(\alpha)b(\alpha)s(\alpha)\,d\alpha + O(\varepsilon^2), \end{aligned} \tag{7}$$

where we used the assumption that $k(A_1) = k'(A_1) = 0$. Since $s(a) = \exp(-\int_0^a lb(\alpha)\,d\alpha)$ depends on l this provides an implicit equation in l. The last approximation holds only if k, b, and hence s are well behaved so that at least the second derivative is bounded.

The equation has exactly one positive solution $l = l^* > 0$. When this l^* is known it is straight forward to compute the equilibrium values of $s^*(a)$ and $i^*(a)$. Still following the method of (Greenhalgh, 1988) and others we determine the local stability of (s^*, i^*) by trying separable perturbations of the form $s(a,t) = s^*(a) + \mathbf{s}(a)e^{pt}$, $i(a,t) = i^*(a) + \mathbf{i}(a)e^{pt}$, and $\lambda(a,t) = \lambda^*(a) + \theta b(a)e^{pt}$. Neglecting higher order terms we obtain a system of ordinary differential equations

$$\begin{aligned}
\frac{d\mathbf{s}}{da} &= -b(a)\theta s^*(a) - (l^* b(a) + p)\mathbf{s} \\
\frac{d\mathbf{i}}{da} &= b(a)\theta s^*(a) + (l^* b(a) + p)\mathbf{s} - (\frac{1}{\varepsilon} + p)\mathbf{i} \\
\theta &= \frac{c}{\varepsilon}\int_0^{A_1} k(\alpha)\mathbf{i}(\alpha)\,d\alpha
\end{aligned}$$

with initial conditions $\mathbf{s}(0) = \mathbf{i}(0) = 0$.

Using the same procedure as above, the stability equation becomes

$$\begin{aligned}
\theta &= \frac{c}{\varepsilon}\int_0^{A_1} k(a)\mathbf{i}(a)\,da \\
&= \frac{c}{\varepsilon}\int_0^{A_1} k(a)\epsilon^{-(\frac{1}{\varepsilon}+p)a}\int_0^a (\theta b(\alpha)s^*(\alpha) + l^* b(\alpha)\mathbf{s}(\alpha))\, e^{(\frac{1}{\varepsilon}+p)\alpha}\,d\alpha\,da \\
&= \frac{c}{\varepsilon}\int_0^{A_1}\int_\alpha^{A_1} k(a)\epsilon^{(\frac{1}{\varepsilon}+p)(\alpha-a)} \\
&\qquad \times \left(\theta b(\alpha)s^*(\alpha) - l^* b(\alpha)s^*(\alpha)\int_0^\alpha \theta b(\alpha')\epsilon^{p(\alpha'-\alpha)}\,d\alpha'\right) da\,d\alpha \\
&= \frac{c}{\varepsilon}\int_0^{A_1}\left(k(\alpha)\frac{\varepsilon}{1+\varepsilon p} + k'(\alpha)\frac{\varepsilon^2}{(1+\varepsilon p)^2}\right) \\
&\qquad \times \left(1 - l^*\int_0^\alpha b(\alpha')e^{p(\alpha'-\alpha)}\,d\alpha'\right)\theta b(\alpha)s^*(\alpha)\,d\alpha \\
&\quad + \frac{c}{\varepsilon}\frac{\varepsilon^2}{(1+\varepsilon p)^2}\int_0^{A_1}\int_\alpha^{A_1} k''(a)e^{(\frac{1}{\varepsilon}+p)(\alpha-a)}\,(\theta b(\alpha)s^*(\alpha) + l^* b(\alpha)\mathbf{s}(\alpha))\,da\,d\alpha
\end{aligned} \tag{8}$$

For $\theta \neq 0$ this yields an equation in p allowing us to identify the possible perturbations.

3 Approximations of the characteristic roots

The general stability equation (8) is hard to analyze so we will now utilize our assumption that $\varepsilon \ll 1$ to allow for a small parameter simplification of (8). Multiplying through by $(1+\varepsilon p)/\theta$ and using the equilibrium condition (7) to simplify the expression, the equation reduces to

$$\begin{aligned} \varepsilon p &= -c\int_0^{A_1} k(a)b(a)s^*(a)l^* \int_0^a b(\alpha)e^{-p(a-\alpha)}\,d\alpha\,da \\ &\quad +c\int_0^{A_1} k'(a)b(a)s^*(a)\left(\frac{\varepsilon}{1+\varepsilon p}-\varepsilon-\frac{\varepsilon l^*}{1+\varepsilon p}\int_0^a b(\alpha)e^{-p(a-\alpha)}\,d\alpha\right)da \\ &\quad +O(\frac{\varepsilon^2}{(1+\varepsilon p)^2})+O(\varepsilon^2). \end{aligned} \tag{9}$$

The terms involving $(1+\varepsilon p)^{-1}$ blow up for $p=-1/\varepsilon$ but roots near $-1/\varepsilon$ are not important for the stability and will not be discussed here. Using underdetermined gauges of the form $|p| = h\varepsilon^q$, we find that for $q \geq 0$ (9) reduces to

$$0 = c\int_0^{A_1} k(a)b(a)s^*(a)l^* \int_0^a b(\alpha)e^{-p(a-\alpha)}\,d\alpha\,da + O(\varepsilon). \tag{10}$$

In the singular case $q<0$ we may approximate (9) by

$$\begin{aligned} \varepsilon p &= -\frac{c}{p}\int_0^{A_1} l^*k(a)b(a)^2s^*(a)\,da \\ &\quad +\frac{c}{p}\int_0^{A_1} l^*k(a)b(a)s^*(a)\left(b(0)\epsilon^{-pa}+\frac{1}{p}b'(a)-\frac{1}{p}b'(0)\epsilon^{-pa}\right)da \\ &\quad +O(\frac{\varepsilon}{p(1+\varepsilon p)})+O(\frac{\varepsilon^2}{(1+\varepsilon p)^2})+O(\frac{\varepsilon^2 p}{(1+\varepsilon p)})+O(\varepsilon^3). \end{aligned}$$

Only the term εp can balance the first term on the right hand side of the equation so the only type of roots with $q<0$ is $|p| = h/\sqrt{\varepsilon}$. To the first order we get

$$p = \pm i\omega/\sqrt{\varepsilon}$$

where

$$\omega^2 = c\int_0^{A_1} l^*k(a)b(a)^2s^*(a)\,da. \tag{11}$$

Since the first order term is purely imaginary we need second order terms to determine the stability of the of the roots and find for $p = \mathrm{i}\,\omega/\sqrt{\varepsilon} + \gamma + O(\sqrt{\varepsilon})$

$$\gamma = -\frac{c}{2\omega^2}\left(l^* k(0) b(0)^2 s^*(0) + \int_0^{A_1} l^* k(a) b(a) s^*(a) b'(a)\, da\right). \qquad (12)$$

Assuming that all dominant roots of (9) are of the form $|p| = h\varepsilon^q$, (10) and (11)-(12) determine the dominant roots of the stability equation and hence the local stability of the endemic equilibrium. I have not been able to obtain general results about these roots but if $b(a)$ and $\tilde{b}(a)$ are both constant then all roots have negative real part and if in addition the host life spans follow a gamma distribution, then the roots $p = \mathrm{i}\,\omega/\sqrt{\varepsilon}$ dominate (Andreasen, 1992).

In the stability equations, the distribution

$$g(a) = cs^*(a) b(a) k(a)$$

plays a significant role. The quantity $g(a)$ may be interpreted as the contribution of individuals of age a to disease transmission at equilibrium, just like $cb(a)k(a)$ gives the contribution of individuals of age a to disease transmission in a susceptible population in the expression for R_0. By (7) $g(a)$ integrates to unity.

The angular frequency ω may now be written as

$$\omega = \sqrt{l^* \bar{b}}$$

where $\bar{b}$ is the average of $b(a)$ with respect to $g(a)$. Here the effect of the age structure on the period splits into two components, a direct effect through $\bar{b}$ and an indirect effect through changes in the force of infection l^*.

The characteristic roots p may be compared with those of the uniform SIR-model without age structure

$$p_u = -\frac{c}{2} \pm \sqrt{(l^* - 1)^2/2 - l^*/\varepsilon} = -\frac{c}{2} \pm \mathrm{i}\sqrt{l^*/\varepsilon} + O(\sqrt{\varepsilon}).$$

Clearly the order $\sqrt{\varepsilon}$-roots (11)-(12) correspond to p_u. Since $\bar{b} = O(1)$ the order $\sqrt{\varepsilon}$-roots give rise to oscillations with a period on the same order of magnitude as p_u. For biologically reasonable parameters this yields oscillation

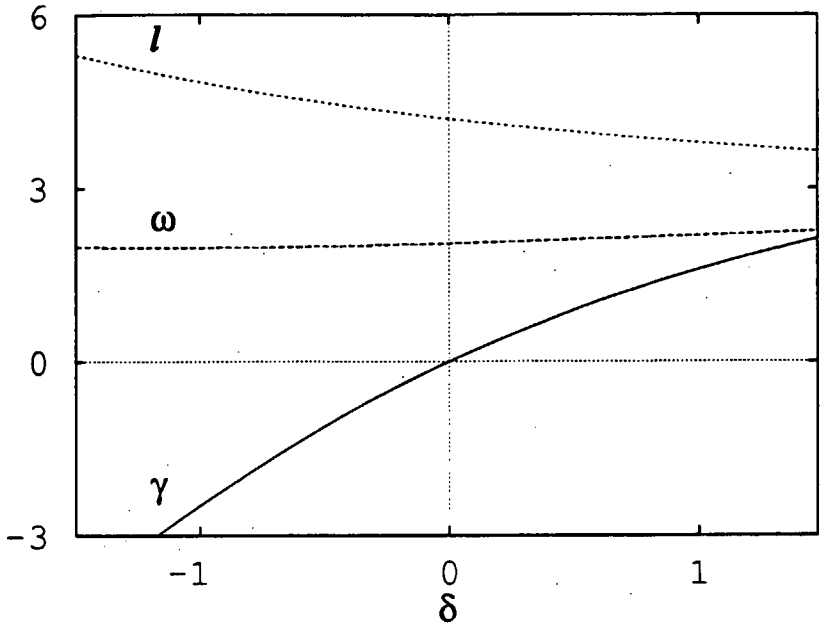

Figure 1: Force of infection $lb(a)$ and dominant eigenvalue $\gamma + i\omega/\sqrt{\varepsilon}$ for the endemic equilibrium of the age-structured SIR-model with proportionate mixing and contact rates determined by (13–14) while $R_0 \approx c = 4$. The slope of the age-dependent disease susceptibility is $-\delta$, hence the equilibrium is unstable when disease susceptibility decreases with age.

on the order of a few years (Anderson and May, 1991). The order 1-roots (10) with their period on the order of host life expectancy $L \approx 75$ yrs. arise from the age structure and have no counterpart in the uniform model. The transition from a uniform population to an age-structured population is discussed in more detail in (Andreasen, 1992).

4 Numerical example

The stability conditions (10) and (11)-(12) depend on the shape of k and b. We will not attempt a general investigation of the stability conditions but

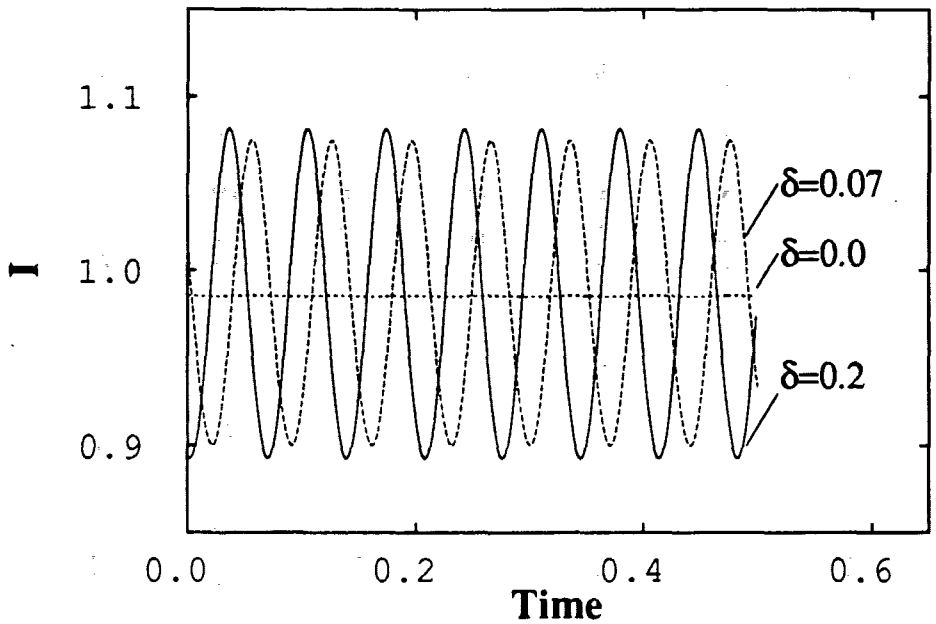

Figure 2: Sustained oscillations in the prevalence $I = \int_0^{A_1} i(a,t)\,da$ of a disease with SIR-dynamics and proportionate mixing as specified by (13–14). The transmission potential is $R_0 \approx c = 4$, while the infectious period is $\varepsilon = 5\times 10^{-4}$ units of host life span. Time is measured in units of host life span L, see text. With $L \approx 70$ yrs. the period of the oscillations is about 5 yrs. When the parameter δ is positive, disease susceptibility decreases with age.

observe that instability can occur if b varies. As a theoretical example – with no biological justification – assume that

$$k(a) = ae^{-1/(1-a)}/K \qquad 0 < a < 1 \tag{13}$$

$$b(a) = -\delta a + (1 - \tfrac{1}{2}\delta), \tag{14}$$

where $-2 < \delta < 2$ and $K = \int_0^1 a\exp(-1/(1-a))\,da$.

Using (7) the real part γ of the order $\varepsilon^{-1/2}$ roots simplifies to

$$\gamma = \frac{c\delta l'}{2\omega^2}$$

so γ changes sign with the sign of δ and a pair of complex eigenvalues passes through the imaginary axis at $\delta = 0$. It is not hard to see that all roots of (10) have negative real part at $\delta = 0$ - and hence by continuity that the roots will remain in the negative half plane for some interval $(0; \delta_1)$ (Andreasen, 1992). Since only one pair of eigenvalues changes sign we expect that a Hopf-type bifurcation occurs (Figure 1).

Numerical simulations of the model are found by solving along characteristics using an implicit first order scheme similar to the one proposed by Ianelli et al (1992). The algorithm is highly damped and step sizes as small as $\Delta a = \Delta t = 1/134400$ are necessary to obtain solutions for $\varepsilon = 5 \times 10^{-4}$. As expected the solutions indicate that a stable limit cycle appears for $\delta > 0 + O(\varepsilon)$ suggesting a supercritical Hopf bifurcation (Figure 2).

5 Discussion

Using the ratio of infectious period to the host life span as a small parameter, perturbation analysis of the stability equation for the endemic equilibrium shows that the spectrum has two dominant components: one near the origin and one with large imaginary part. The relative position and sign of the real part of these roots are not known in general. If disease transmission is independent of age then both types of roots have negative real part and if in addition host life span follows a gamma distribution, then the roots with large imaginary part dominate (Andreasen, 1992). A generalization of these results to the proportionate mixing case would be nice but is not obvious.

The main result in this paper is that age-dependent disease transmission can lead to sustained oscillations with the characteristic period of a few years known from many childhood diseases. When disease transmission follows proportionate mixing, sustained oscillations can occur only if disease susceptibility is 'on the average' decreasing with age. Biologically this seems to be an unlikely mechanism for the maintenance of recurrent epidemics as much empirical evidence shows that the experienced force of infection increases with age - at least until an age where most individuals have been infected (Anderson and May, 1985).

Our stability analysis relied heavily on the assumption of proportionate mixing but it may be possible to generalize the results to other transmission patterns. Anderson and May (1985, p. 372) propose a two age class model where the force of infection in age class i is determined by

$$\Lambda_i = \frac{b_{i1}}{\varepsilon}\bar{I}_1 + \frac{b_{i2}}{\varepsilon}\bar{I}_2. \qquad i = 1,2$$

Here $\bar{I}_1 = \int_0^{A_c} I(a,t)\,da$ and $\bar{I}_2 = \int_{A_c}^1 I(a,t)\,da$ give the number of infectious individuals in the two age classes. If $\Delta = b_{11}b_{22} - b_{12}b_{21} \neq 0$ disease transmission is not of the proportionate mixing type. Extension of the method of Anderson and May (1985) to the first order separable perturbations yields a stability equation that is amendable to the limiting procedure of this paper. Under the assumption of a short infectious period, the roots of the stability equation for an endemic equilibrium show the same structure as for proportionate mixing, i.e. there are two types of dominant roots, one type near the origin and one type of the form $p = \gamma + \mathrm{i}\,\omega/\sqrt{\varepsilon}$ with

$$\omega^2 = \frac{b_{11}\Lambda_1\bar{S}_1 + b_{22}\Lambda_2\bar{S}_2 - \Delta\bar{S}_1\bar{S}_2(\Lambda_1 + \Lambda_2)}{1 - \Delta\bar{S}_1\bar{S}_2},$$

where $\bar{S}_i$ is defined in the same way as $\bar{I}_i$. The second order term γ is sensitive to the discretization of the age structure and is too messy to allow for analysis, but the result suggests that the occurrence of roots with large imaginary part may be a general feature of SIR-models with short infectious period. Similarly one finds that the endemic equilibrium of SEIR-models with short latent and infectious periods have roots with large imaginary part.

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101/85 "EXTENDED MOMENTUM THEORY FOR WINDMILLS IN PERTURBATIVE FORM".
Af: Ganesh Sengupta.

102/85 OPSTILLING OG ANALYSE AF MATEMATISKE MODELLER, BELYST VED MODELLER OVER KØERS FODEROPTAGELSE OG - OMSÆTNING".
Projektrapport af: Lis Eilertzen, Kirsten Habekost, Lill Røn og Susanne Stender.
Vejleder: Klaus Grünbaum.

103/85 "ØDSLE KOLDKRIGERE OG VIDENSKABENS LYSE IDEER".
Projektrapport af: Niels Ole Dam og Kurt Jensen.
Vejleder: Bent Sørensen.

104/85 "ANALOGREGNEMASKINEN OG LORENZLIGNINGER".
Af: Jens Jæger.

105/85 "THE FREQUENCY DEPENDENCE OF THE SPECIFIC HEAT AT THE GLASS REANSITION".
Af: Tage Christensen.

"A SIMPLE MODEL AF AC HOPPING CONDUCTIVITY".
Af: Jeppe C. Dyre.
Contributions to the Third International Conference on the Structure of Non - Crystalline Materials held in Grenoble July 1985.

106/85 "QUANTUM THEORY OF EXTENDED PARTICLES".
Af: Bent Sørensen.

107/85 "EN MYG GØR INGEN EPIDEMI",
- flodblindhed som eksempel på matematisk modellering af et epidemiologisk problem.
Projektrapport af: Per Hedegård Andersen, Lars Boye, CarstenHolst Jensen, Else Marie Pedersen og Erling Møller Pedersen.
Vejleder: Jesper Larsen.

108/85 "APPLICATIONS AND MODELLING IN THE MATEMATICS CURRICULUM" - state and trends -
Af: Mogens Niss.

109/85 "COX I STUDIETIDEN" - Cox's regressionsmodel anvendt på studenteroplysninger fra RUC.
Projektrapport af: Mikael Wennerberg Johansen, Poul Katler og Torben J. Andreasen.
Vejleder: Jørgen Larsen.

110/85 "PLANNING FOR SECURITY".
Af: Bent Sørensen

111/85 JORDEN RUNDT PÅ FLADE KORT".
Projektrapport af: Birgit Andresen, Beatriz Quinones og Jimmy Staal.
Vejleder: Mogens Niss.

112/85 "VIDENSKABELIGGØRELSE AF DANSK TEKNOLOGISK INNOVATION FREM TIL 1950 - BELYST VED EKSEMPLER".
Projektrapport af: Erik Odgaard Gade, Hans Hedal, Frank C. Ludvigsen, Annette Post Nielsen og Finn Physant.
Vejleder: Claus Bryld og Bent C. Jørgensen.

113/85 "DESUSPENSION OF SPLITTING ELLIPTIC SYMBOLS II".
Af: Bernhelm Booss og Krzysztof Wojciechowski.

114/85 "ANVENDELSE AF GRAFISKE METODER TIL ANALYSE AF KONTIGENSTABELLER".
Projektrapport af: Lone Biilmann, Ole R. Jensen og Anne-Lise von Moos.
Vejleder: Jørgen Larsen.

115/85 "MATEMATIKKENS UDVIKLING OP TIL RENÆSSANCEN".
Af: Mogens Niss.

116/85 "A PHENOMENOLOGICAL MODEL FOR THE MEYER-NELDEL RULE".
Af: Jeppe C. Dyre.

117/85 "KRAFT & FJERNVARMEOPTIMERING"
Af: Jacob Mørch Pedersen.
Vejleder: Bent Sørensen

118/85 "TILFÆLDIGHEDEN OG NØDVENDIGHEDEN IFØLGE PEIRCE OG FYSIKKEN".
Af: Peder Voetmann Christiansen

119/86 "DET ER GANSKE VIST - - EUKLIDS FEMTE POSTULAT KUNNE NOK SKABE RØRE I ANDEDAMMEN".
Af: Iben Maj Christiansen
Vejleder: Mogens Niss.

120/86 "ET ANTAL STATISTISKE STANDARDMODELLER".
Af: Jørgen Larsen

121/86 "SIMULATION I KONTINUERT TID".
Af: Peder Voetmann Christiansen.

122/86 "ON THE MECHANISM OF GLASS IONIC CONDUCTIVITY".
Af: Jeppe C. Dyre.

123/86 "GYMNASIEFYSIKKEN OG DEN STORE VERDEN".
Fysiklærerforeningen, IMFUFA, RUC.

124/86 "OPGAVESAMLING I MATEMATIK".
Samtlige opgaver stillet i tiden 1974-jan. 1986.

125/86 "UVBY,β - systemet - en effektiv fotometrisk spektralklassifikation af B-,A- og F-stjerner".
Projektrapport af: Birger Lundgren.

126/86 "OM UDVIKLINGEN AF DEN SPECIELLE RELATIVITETSTEORI".
Projektrapport af: Lise Odgaard & Linda Szkotak Jensen
Vejledere: Karin Beyer & Stig Andur Pedersen.

127/86 "GALOIS' BIDRAG TIL UDVIKLINGEN AF DEN ABSTRAKTE ALGEBRA".
Projektrapport af: Pernille Sand, Heine Larsen & Lars Frandsen.
Vejleder: Mogens Niss.

128/86 "SMÅKRYB" - om ikke-standard analyse.
Projektrapport af: Niels Jørgensen & Mikael Klintorp.
Vejleder: Jeppe Dyre.

129/86 "PHYSICS IN SOCIETY"
Lecture Notes 1983 (1986)
Af: Bent Sørensen

130/86 "Studies in Wind Power"
Af: Bent Sørensen

131/86 "FYSIK OG SAMFUND" - Et integreret fysik/historieprojekt om naturanskuelsens historiske udvikling og dens samfundsmæssige betingethed.
Projektrapport af: Jakob Heckscher, Søren Brønd, Andy Wierød.
Vejledere: Jens Høyrup, Jørgen Vogelius, Jens Højgaard Jensen.

132/86 "FYSIK OG DANNELSE"
Projektrapport af: Søren Brønd, Andy Wierød.
Vejledere: Karin Beyer, Jørgen Vogelius.

133/86 "CHERNOBYL ACCIDENT: ASSESSING THE DATA.
ENERGY SERIES NO. 15.
AF: Bent Sørensen.

134/87 "THE D.C. AND THE A.C. ELECTRICAL TRANSPORT IN AsSeTe SYSTEMS"
Authors: M.B.El-Den, N.B.Olsen, Ib Høst Pedersen, Petr Viščor

135/87 "INTUITIONISTISK MATEMATIKS METODER OG ERKENDELSESTEORETISKE FORUDSÆTNINGER"
MASTEMATIKSPECIALE: Claus Larsen
Vejledere: Anton Jensen og Stig Andur Pedersen

136/87 "Mystisk og naturlig filosofi: En skitse af kristendommens første og andet møde med græsk filosofi"
Projektrapport af Frank Colding Ludvigsen
Vejledere: Historie: Ib Thiersen
Fysik: Jens Højgaard Jensen

137/87 "HOPMODELLER FOR ELEKTRISK LEDNING I UORDNEDE FASTE STOFFER" - Resume af licentiatafhandling
Af: Jeppe Dyre
Vejledere: Niels Boye Olsen og Peder Voetmann Christiansen.

138/87 "JOSEPHSON EFFECT AND CIRCLE MAP."

Paper presented at The International Workshop on Teaching Nonlinear Phenomena at Universities and Schools, "Chaos in Education". Balaton, Hungary, 26 April-2 May 1987.

By: Peder Voetmann Christiansen

139/87 "Machbarkeit nichtbeherrschbarer Technik durch Fortschritte in der Erkennbarkeit der Natur"

Af: Bernhelm Booss-Bavnbek
Martin Bohle-Carbonell

140/87 "ON THE TOPOLOGY OF SPACES OF HOLOMORPHIC MAPS"

By: Jens Gravesen

141/87 "RADIOMETERS UDVIKLING AF BLODGASAPPARATUR - ET TEKNOLOGIHISTORISK PROJEKT"

Projektrapport af Finn C. Physant

Vejleder: Ib Thiersen

142/87 "The Calderón Projektor for Operators With Splitting Elliptic Symbols"

by: Bernhelm Booss-Bavnbek og
Krzysztof P. Wojciechowski

143/87 "Kursusmateriale til Matematik på NAT-BAS"

af: Mogens Brun Heefelt

144/87 "Context and Non-Locality - A Peircean Approach

Paper presented at the Symposium on the Foundations of Modern Physics The Copenhagen Interpretation 60 Years after the Como Lecture. Joensuu, Finland, 6 - 8 august 1987.

By: Peder Voetmann Christiansen

145/87 "AIMS AND SCOPE OF APPLICATIONS AND MODELLING IN MATHEMATICS CURRICULA"

Manuscript of a plenary lecture delivered at ICMTA 3, Kassel, FRG 8.-11.9.1987

By: Mogens Niss

146/87 "BESTEMMELSE AF BULKRESISTIVITETEN I SILICIUM"

- en ny frekvensbaseret målemetode.

Fysikspeciale af Jan Vedde

Vejledere: Niels Boye Olsen & Petr Viščor

147/87 "Rapport om BIS på NAT-BAS"

redigeret af: Mogens Brun Heefelt

148/87 "Naturvidenskabsundervisning med Samfundsperspektiv"

af: Peter Colding-Jørgensen DLH
Albert Chr. Paulsen

149/87 "In-Situ Measurements of the density of amorphous germanium prepared in ultra high vacuum"

by: Petr Viščor

150/87 "Structure and the Existence of the first sharp diffraction peak in amorphous germanium prepared in UHV and measured in-situ"

by: Petr Viščor

151/87 "DYNAMISK PROGRAMMERING"

Matematikprojekt af:
Birgit Andresen, Keld Nielsen og Jimmy Staal

Vejleder: Mogens Niss

152/87 "PSEUDO-DIFFERENTIAL PROJECTIONS AND THE TOPOLOGY OF CERTAIN SPACES OF ELLIPTIC BOUNDARY VALUE PROBLEMS"

by: Bernhelm Booss-Bavnbek
Krzysztof P. Wojciechowski

153/88 "HALVLEDERTEKNOLOGIENS UDVIKLING MELLEM MILITÆRE OG CIVILE KRÆFTER"

Et eksempel på humanistisk teknologihistorie
Historiespeciale

Af: Hans Hedal

Vejleder: Ib Thiersen

154/88 "MASTER EQUATION APPROACH TO VISCOUS LIQUIDS AND THE GLASS TRANSITION"

By: Jeppe Dyre

155/88 "A NOTE ON THE ACTION OF THE POISSON SOLUTION OPERATOR TO THE DIRICHLET PROBLEM FOR A FORMALLY SELFADJOINT DIFFERENTIAL OPERATOR"

by: Michael Pedersen

156/88 "THE RANDOM FREE ENERGY BARRIER MODEL FOR AC CONDUCTION IN DISORDERED SOLIDS"

by: Jeppe C. Dyre

157/88 " STABILIZATION OF PARTIAL DIFFERENTIAL EQUATIONS BY FINITE DIMENSIONAL BOUNDARY FEEDBACK CONTROL: A pseudo-differential approach."

by: Michael Pedersen

158/88 "UNIFIED FORMALISM FOR EXCESS CURRENT NOISE IN RANDOM WALK MODELS"

by: Jeppe Dyre

159/88 "STUDIES IN SOLAR ENERGY"

by: Bent Sørensen

160/88 "LOOP GROUPS AND INSTANTONS IN DIMENSION TWO"

by: Jens Gravesen

161/88 "PSEUDO-DIFFERENTIAL PERTURBATIONS AND STABILIZATION OF DISTRIBUTED PARAMETER SYSTEMS:

Dirichlet feedback control problems"

by: Michael Pedersen

162/88 "PIGER & FYSIK - OG MEGET MERE"

AF: Karin Beyer, Sussanne Blegaa, Birthe Olsen,
Jette Reich , Mette Vedelsby

163/88 "EN MATEMATISK MODEL TIL BESTEMMELSE AF PERMEABILITETEN FOR BLOD-NETHINDE-BARRIEREN"

Af: Finn Langberg, Michael Jarden, Lars Frellesen

Vejleder: Jesper Larsen

164/88 "Vurdering af matematisk teknologi
Technology Assessment
Technikfolgenabschätzung"

Af: Bernhelm Booss-Bavnbek, Glen Pate med
Martin Bohle-Carbonell og Jens Højgaard Jensen

165/88 "COMPLEX STRUCTURES IN THE NASH-MOSER CATEGORY"

by: Jens Gravesen

166/88 "Grundbegreber i Sandsynligheds-regningen"

Af: Jørgen Larsen

167a/88 "BASISSTATISTIK 1. Diskrete modeller"

Af: Jørgen Larsen

167b/88 "BASISSTATISTIK 2. Kontinuerte modeller"

Af: Jørgen Larsen

168/88 "OVERFLADEN AF PLANETEN MARS"
Laboratorie-simulering og MARS-analoger undersøgt ved Mössbauerspektroskopi.

Fysikspeciale af:

Birger Lundgren

Vejleder: Jens Martin Knudsen
Fys.Lab./HCØ

169/88 "CHARLES S. PEIRCE: MURSTEN OG MØRTEL TIL EN METAFYSIK."

Fem artikler fra tidsskriftet "The Monist" 1891-93.

Introduktion og oversættelse:

Peder Voetmann Christiansen

170/88 "OPGAVESAMLING I MATEMATIK"

Samtlige opgaver stillet i tiden 1974 - juni 1988

171/88 "The Dirac Equation with Light-Cone Data"

af: Johnny Tom Ottesen

172/88 "FYSIK OG VIRKELIGHED"

Kvantemekanikkens grundlagsproblem i gymnasiet.

Fysikprojekt af:

Erik Lund og Kurt Jensen

Vejledere: Albert Chr. Paulsen og Peder Voetmann Christiansen

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173/89 "NUMERISKE ALGORITMER"

af: Mogens Brun Heefelt

174/89 " GRAFISK FREMSTILLING AF FRAKTALER OG KAOS"

af: Peder Voetmann Christiansen

175/89 " AN ELEMENTARY ANALYSIS OF THE TIME DEPENDENT SPECTRUM OF THE NON-STATONARY SOLUTION TO THE OPERATOR RICCATI EQUATION

af: Michael Pedersen

176/89 " A MAXIUM ENTROPY ANSATZ FOR NONLINEAR RESPONSE THEORY"

af : Jeppe Dyre

177/89 "HVAD SKAL ADAM STÅ MODEL TIL"

af: Morten Andersen, Ulla Engström, Thomas Gravesen, Nanna Lund, Pia Madsen, Dina Rawat, Peter Torstensen

Vejleder: Mogens Brun Heefelt

178/89 "BIOSYNTESEN AF PENICILLIN - en matematisk model"

af: Ulla Eghave Rasmussen, Hans Oxvang Mortensen, Michael Jarden

vejleder i matematik: Jesper Larsen
biologi: Erling Lauridsen

179a/89 "LÆRERVEJLEDNING M.M. til et eksperimentelt forløb om kaos"

af: Andy Wierød, Søren Brønd og Jimmy Staal

Vejledere: Peder Voetmann Christiansen
Karin Beyer

179b/89 "ELEVHÆFTE: Noter til et eksperimentelt kursus om kaos"

af: Andy Wierød, Søren Brønd og Jimmy Staal

Vejledere: Peder Voetmann Christiansen
Karin Beyer

180/89 "KAOS I FYSISKE SYSTEMER eksemplificeret ved torsions- og dobbeltpendul".

af: Andy Wierød, Søren Brønd og Jimmy Staal
Vejleder: Peder Voetmann Christiansen

181/89 "A ZERO-PARAMETER CONSTITUTIVE RELATION FOR PURE SHEAR VISCOELASTICITY"

by: Jeppe Dyre

183/89 "MATEMATICAL PROBLEM SOLVING, MODELLING. APPLICATIONS AND LINKS TO OTHER SUBJECTS - State. trends and issues in mathematics instruction

by: WERNER BLUM, Kassel (FRG) og
MOGENS NISS, Roskilde (Denmark)

184/89 "En metode til bestemmelse af den frekvensafhængige varmefylde af en underafkølet væske ved glasovergangen"

af: Tage Emil Christensen

185/90 "EN NÆSTEN PERIODISK HISTORIE"

Et matematisk projekt

af: Steen Grode og Thomas Jessen

Vejleder: Jacob Jacobsen

186/90 "RITUAL OG RATIONALITET i videnskabers udvikling"

redigeret af Arne Jakobsen og Stig Andur Pedersen

187/90 "RSA - et kryptografisk system"

af: Annemette Sofie Olufsen, Lars Frellesen og Ole Møller Nielsen

Vejledere: Michael Pedersen og Finn Munk

188/90 "FERMICONDENSATION - AN ALMOST IDEAL GLASS TRANSITION"

by: Jeppe Dyre

189/90 "DATAMATER I MATEMATIKUNDERVISNINGEN PÅ GYMNASIET OG HØJERE LÆREANSTALTER

af: Finn Langberg

190/90 "FIVE REQUIREMENTS FOR AN APPROXIMATE NONLINEAR RESPONSE THEORY"

by: Jeppe Dyre

191/90 "MOORE COHOMOLOGY, PRINCIPAL BUNDLES AND ACTIONS OF GROUPS ON C*-ALGEBRAS"

by: Iain Raeburn and Dana P. Williams

192/90 "Age-dependent host mortality in the dynamics of endemic infectious diseases and
SIR-models of the epidemiology and natural selection of co-circulating influenza virus with partial cross-immunity"

by: Viggo Andreasen

193/90 "Causal and Diagnostic Reasoning"

by: Stig Andur Pedersen

194a/90 "DETERMINISTISK KAOS"
Projektrapport af : Frank Olsen

194b/90 "DETERMINISTISK KAOS"
Kørselsrapport

Projektrapport af: Frank Olsen

195/90 "STADIER PÅ PARADIGMETS VEJ"
Et projekt om den videnskabelige udvikling der førte til dannelse af kvantemekanikken.

Projektrapport for 1. modul på fysikuddannelsen, skrevet af:

Anja Boisen, Thomas Hougård, Anders Gorm Larsen, Nicolai Ryge.

Vejleder: Peder Voetmann Christiansen

196/90 "ER KAOS NØDVENDIGT?"

- en projektrapport om kaos' paradigmatiske status i fysikken.

af: Johannes K. Nielsen, Jimmy Staal og Peter Bøggild

Vejleder: Peder Voetmann Christiansen

197/90 "Kontrafaktiske konditionaler i HOL

af: Jesper Voetmann, Hans Oxvang Mortensen og Aleksander Høst-Madsen

Vejleder: Stig Andur Pedersen

198/90 "Metal-Isolator-Metal systemer"

Speciale

af: Frank Olsen

199/90 "SPREDT FÆGTNING" Artikelsamling

af: Jens Højgaard Jensen

200/90 "LINEÆR ALGEBRA OG ANALYSE"

Noter til den naturvidenskabelige basisuddannelse.

af: Mogens Niss

201/90 **"Undersøgelse af atomare korrelationer i amorfe stoffer ved røntgendiffraktion"**

af: Karen Birkelund og Klaus Dahl Jensen

Vejledere: Petr Viščor, Ole Bakander

202/90 **"TEGN OG KVANTER"**
Foredrag og artikler, 1971-90.

af: Peder Voetmann Christiansen

203/90 **"OPGAVESAMLING I MATEMATIK" 1974-1990**

afløser tekst 170/88

204/91 "ERKENDELSE OG KVANTEMEKANIK"

et Breddemodul Fysik Projekt

af: Thomas Jessen

Vejleder: Petr Viščor

205/91 "PEIRCE'S LOGIC OF VAGUENESS"

by: Claudine Engel-Tiercelin
Department of Philosophy
Université de Paris-1
(Panthéon-Sorbonne)

206a+b/91 "GERMANIUMBEAMANALYSE SAMT A - GE TYNDFILMS ELEKTRISKE EGENSKABER"

Eksperimentelt Fysikspeciale

af: Jeanne Linda Mortensen

og Annette Post Nielsen

Vejleder: Petr Viščor

207/91 "SOME REMARKS ON AC CONDUCTION IN DISORDERED SOLIDS"

by: Jeppe C. Dyre

208/91 "LANGEVIN MODELS FOR SHEAR STRESS FLUCTUATIONS IN FLOWS OF VISCO-ELASTIC LIQUIDS"

by: Jeppe C. Dyre

209/91 "LORENZ GUIDE" Kompendium til den danske fysiker Ludvig Lorenz, 1829-91.

af: Helge Kragh

210/91 "Global Dimension, Tower of Algebras, and Jones Index of Split Seperable Subalgebras with Unitality Condition.

by: Lars Kadison

211/91 "I SANDHEDENS TJENESTE"

- historien bag teorien for de komplekse tal.

af: Lise Arleth, Charlotte Gjerrild, Jane Hansen, Linda Kyndlev, Anne Charlotte Nilsson, Kamma Tulinius.

Vejledere: Jesper Larsen og Bernhelm Booss-Bavnbek

212/91 "Cyclic Homology of Triangular Matrix Algebras"

by: Lars Kadison

213/91 "Disease-induced natural selection in a diploid host

by: Viggo Andreasen and Freddy B. Christiansen

214|91 "Halløj i æteren" - om elektromagnetisme. Oplæg til undervisningsmateriale i gymnasiet.

Af: Nils Kruse, Peter Gastrup, Kristian Hoppe, Jeppe Guldager

Vejledere: Petr Viscor, Hans Hedal

215|91 "Physics and Technology of Metal-Insulator-Metal thin film structures used as planar electron emitters

by: A.Delong, M.Drsticka, K.Hladil, V.Kolarik, F.Olsen, P.Pavelka and Petr Viscor.

216|91 "Kvantemekanik på PC'eren"

af: Thomas Jessen

217/92 "Two papers on APPLICATIONS AND MODELLING IN THE MATHEMATICS CURRICULUM"

by: Mogens Niss

218/92 "A Three-Square Theorem"

by: Lars Kadison

219/92 "RUPNOK - stationær strømning i elastiske rør"

af: Anja Boisen, Karen Birkelund, Mette Olufsen

Vejleder: Jesper Larsen

220/92 "Automatisk diagnosticering i digitale kredsløb"

af: Bjørn Christensen, Ole Møller Nielsen

Vejleder: Stig Andur Pedersen

221/92 "A BUNDLE VALUED RADON TRANSFORM, WITH APPLICATIONS TO INVARIANT WAVE EQUATIONS"

by: Thomas P. Branson, Gestur Olafsson and Henrik Schlichtkrull

222/92 On the Representations of some Infinite Dimensional Groups and Algebras Related to Quantum Physics

by: Johnny T. Ottesen

223/92 *THE FUNCTIONAL DETERMINANT*

by: Thomas P. Branson

224/92 *UNIVERSAL AC CONDUCTIVITY OF NON-METALLIC SOLIDS AT LOW TEMPERATURES*

by: Jeppe C. Dyre

225/92 *"HATMODELLEN" Impedansspektroskopi i ultrarent en-krystallinsk silicium*

af: Anja Boisen, Anders Gorm Larsen, Jesper Varmer, Johannes K. Nielsen, Kit R. Hansen, Peter Bøggild og Thomas Hougaard

Vejleder: Petr Viscor

226/92 *"METHODS AND MODELS FOR ESTIMATING THE GLOBAL CIRCULATION OF SELECTED EMISSIONS FROM ENERGY CONVERSION"*

by: Bent Sørensen

227/92 "Computersimulering og fysik"

af: Per M.Hansen, Steffen Holm, Peter Maibom, Mads K. Dall Petersen, Pernille Postgaard, Thomas B.Schrøder, Ivar P. Zeck

Vejleder: Peder Voetmann Christiansen

228/92 "Teknologi og historie"

Fire artikler af:

Mogens Niss, Jens Høyrup, Ib Thiersen, Hans Hedal

229/92 "Masser af information uden betydning"

En diskussion af informationsteorien i Tor Nørretranders' "Mærk Verden" og en skitse til et alternativ basseret på andenordens kybernetik og semiotik.

af: Søren Brier

230/92 "Vinklens tredeling - et klassisk problem"

et matematisk projekt af

Karen Birkelund, Bjørn Christensen

Vejleder: Johnny Ottesen

231A/92 "Elektrondiffusion i silicium - en matematisk model"

af: Jesper Voetmann, Karen Birkelund, Mette Olufsen, Ole Møller Nielsen

Vejledere: Johnny Ottesen, H.B.Hansen

231B/92 "Elektrondiffusion i silicium - en matematisk model" Kildetekster

af: Jesper Voetmann, Karen Birkelund, Mette Olufsen, Ole Møller Nielsen

Vejledere: Johnny Ottesen, H.B.Hansen

232/92 "Undersøgelse om den simultane opdagelse af energiens bevarelse og især eles om de af Mayer, Colding, Joule og Helmholtz udførte arbejder"

af: L.Arleth, G.I.Dybkjær, M.T.Østergård

Vejleder: Dorthe Posselt

233/92 "The effect of age-dependent host mortality on the dynamics of an endemic disease and
Instability in an SIR-model with age-dependent susceptibility

by: Viggo Andreasen